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Chronic General Periodontitis:

"Periodontal Disease" (Pyorrhœa Alveolaris)

BY

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PREFACE.

The following pages treat of the morbid condition of the teeth which is characterized by a slowly progressive destruction of the tooth attachments. The condition has been variously designated, but the term *pyorrhœa alveolaris* has probably been most commonly used and no doubt at one time appeared to be justified by what were considered to be the clinical characteristics of the condition. Recent investigations have added to our knowledge of the causes and development of this condition, and what was regarded as a specific disease by earlier writers is to-day recognized as a suppurative stage of a disease which can now be traced through all its stages from its early beginning as a marginal gingivitis to an advanced suppurative condition of the tooth-sockets.

The outstanding feature of the disease is a chronic infection of the periodontal membrane, and it would seem to be more appropriate therefore to describe the disease as a *chronic general periodontitis*.

The disease has attracted my attention for many years and, from time to time, I have published papers dealing with its various aspects. This book is to a great extent a reprint of those papers and it is being published in the hope that it may be helpful to those who day by day are endeavouring to grapple with this widespread disease.

I desire to take this opportunity of expressing my appreciation of the valuable assistance rendered by several friends in the preparation of this book, and I wish particularly to record my obligations to Mr. W. Lang, who kindly wrote the section on eye lesions and dental sepsis; to Mr. Penfold for his investigation into the relation of the *Entamœba buccalis* to the disease; to Mr. J. Eyre for revising the section dealing with vaccines; to my

colleague, Mr. Norman Bennett, for the section on ionic treatment, and to Mr. H. Darby for much kind help.

For the loan of blocks for the illustrations I wish to express my thanks to Messrs. Longmans, Green and Co., The Royal Society of Medicine, and the Medical Committee of the Royal Dental Hospital.

J. F. Colyer.

May, 1916.

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DENTAL SCHOOL

Chronic General Periodontitis

(Periodontal Disease, Pyorrhœa Alveolaris).

CHAPTER I.

The Macroscopical Anatomy of Chronic General Periodontitis.

BEFORE a description is given of the morbid anatomy of periodontal disease, it is necessary to consider certain aspects of the normal anatomy of the parts involved in the disease, in order that the pathological changes may be more readily appreciated.

Fig. 1 shows the mandible of a female, aged 22. The alveolar process may be regarded as normal. The alveolar process and the teeth sockets are everywhere lined with a fairly dense layer of bone, which appears in the skiagram (fig. 2) as a dark line. On the buccal aspect in the premolar and molar region the margin of the bone is close below the necks of the teeth, and, stretching across from one tooth to another in a straight line, forms the base of a definite triangular space between the approximal aspects of the teeth. In the incisor region the bone does not follow a straight line, but is continued upwards, so that the space between the incisors is considerably less than between the premolars and molars. The margin of the bone presents a regular outline, and the surface of the bone, which is smooth, is pierced here and there with nutrient canals. The appearance of the bone in skiagrams is shown in fig. 2. Between the front teeth the bone has the appearance of a sharp spine, but it passes in a straight line between the back teeth. The outer aspect of the bone shows a

dense layer, the inner part presenting a cancellous appearance. In the maxilla a similar condition obtains.[1]

As age advances there is a gradual tendency for the alveolar process to disappear. In healthy mouths, the gum margin recedes as the alveolar process disappears and a layer of compact

FIG. 1.—Mandible of female, aged 22, with an alveolar process which may be regarded as normal.

FIG. 2.— Skiagrams showing the appearance of the bone in the mandible (fig. 1).

tissue is constantly present over the alveolar process. The skiagrams of an individual aged 30 with normal gums are shown in fig. 3. The muco-periosteum should be firm and pale pink in colour. It should blend with the periodontal membrane at the

[1] The skiagram of the incisors shows a slight break in the compact tissue towards the apex of the spine, but this is due to slight fracture (*post mortem*).

neck of the tooth, the junction being distinguishable by the greater density in the character of the fibrous tissue. The gingival margin of the muco-periosteum is not attached to the tooth on

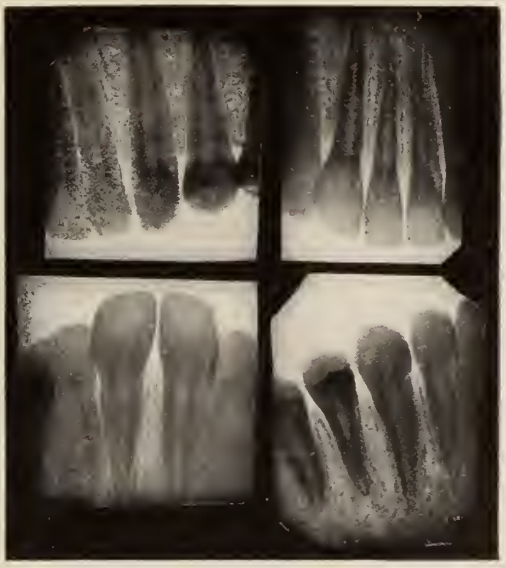

FIG. 3.—Skiagrams of alveolar process of an individual with normal gum.

FIG. 4.—Maxilla which shows slight destruction of the bone in the centre of the interproximal spaces.

the general level of the gum, as the gum falls slightly before reaching the tooth and forms a slight depression around the tooth. This depression is generally termed the "gingival space," but the term "space" is not altogether satisfactory as the soft

tissues actually lie in contact with the surface of the tooth. The floor of the depression is at the neck of the tooth, and consequently with the eruption of the tooth the depth of the depression decreases. The physiology of the "gingival space" is not known.

Clinical experience shows that periodontal disease usually originates in the molar region or around the incisors. When

FIG. 5.—Skiagrams of maxilla shown in fig. 4.
Note that the destruction of the bone has already commenced in the interproximal spaces.

FIG. 6.—Maxilla in which the destruction of the bone has advanced beyond the stage shown in fig. 4.

it originates around the back teeth the initial lesion in the bone is a slight destruction of tissue in the centre of the interproximal spaces as shown in fig. 4, a specimen which has the appearance of being normal unless carefully scrutinized. In the next specimen (fig. 6), the disappearance of the layer of dense tissue and the breach into the cancellous bone indicate that the destruction of the bone has already commenced.

As the destruction of the bone proceeds the outer and inner plates of the alveolar process become involved, but the loss of tissue, as a rule, is greater between the teeth than on the buccal and palatal aspects. This stage is shown in fig. 6.

FIG. 7.—Maxilla showing considerable destruction of the alveolar process.

FIG. 8.—Specimen in which the disease probably commenced around the incisors.

It will be observed that :—

(1) The destruction of bone around the front teeth is considerably less than around the premolars and molars ; and (2) the loss of tissue around the molars is greater than around the premolars.

A further stage of the disease is shown in fig. 7. The bone has so far disappeared as to expose about half of the roots of

the incisors and two-thirds of the roots of the molars. Around the canine and premolars the bone destruction is less advanced.

A specimen in which the disease probably commenced around the incisor teeth is shown in fig. 8. There is some

FIG. 9.—Skiagrams of specimen shown in fig. 8. The extent of the bone lesion and the resistance of the tissue between the central incisors is clearly shown.

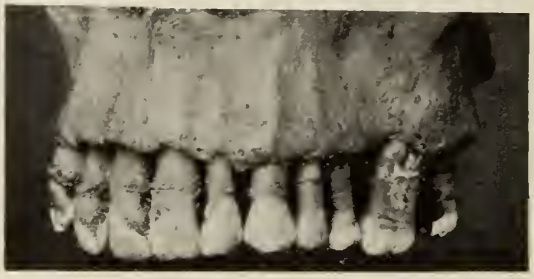

FIG. 10.—This specimen from a male, aged 30, shows the morbid anatomy of an advanced case of periodontal disease.

loss of bone between the teeth, and the roots on the labial aspects are uncovered. The margin of the bone shows the pitted, irregular appearance characteristic of rarefying osteitis. It will be observed that there has been less destruction of bone between the central incisors than between the other front teeth.

An examination of the specimen shows that around the incisors the destruction of bone is more advanced than around the molar teeth. The skiagrams (fig. 9) bring out clearly the extent of the bone lesion and also the resistance of the tissue between the central incisors.

The specimen shown in fig. 10 from a male, aged 30, is an excellent example of the morbid anatomy of an advanced case. The bone around the incisors has almost disappeared, and the

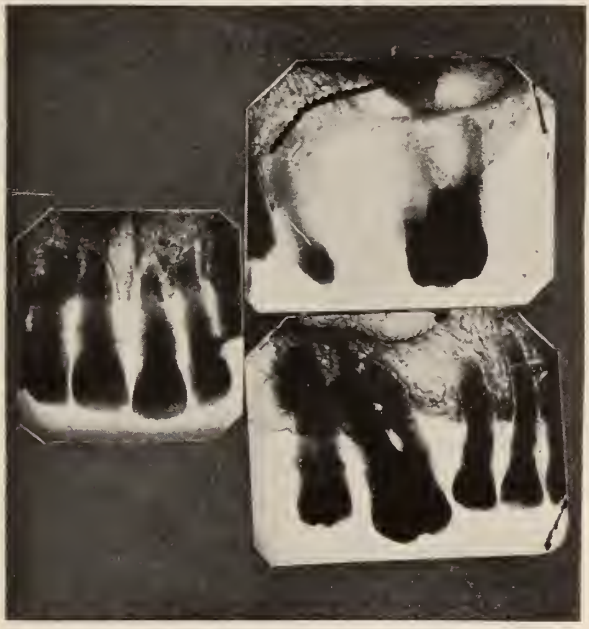

FIG. 11.—Skiagrams of maxillæ shown in fig. 10.

molar teeth are uncovered nearly to their apices. Around the teeth are cup-shaped spaces which are due to the fact that the bone bordering the tooth disappears first. The appearance of the bone shows that there has been a marked rarefying osteitis. Nodular deposits of calculus are present on the teeth, and it will be observed that these are, in the case of the incisors, canines, and premolars, well away from the margin of the bone. The skiagrams of this specimen are shown in fig. 11. The

cup-shaped absorption of the socket is clearly shown in the right first molar of the specimen illustrated in fig. 12. Around the second molar the destruction of the bone has been of a more chronic type, the cup-shaped absorption being absent. The

FIG. 12.—Maxilla showing cup-shaped absorption of the bone around the right first molar.

FIG. 13.—Skiagram of the first and second molars in fig. 12, showing the different degrees of bone destruction around these teeth.

skiagram of these teeth (fig. 13) shows a very distinct difference in appearance. Around the roots of the first molar there is a definite clear area and the dense layer which marks the outline of the normal socket is absent. The bone around the tooth shows definite signs of rarefying osteitis. The bone bordering

the roots of the second molar still retains the dense appearance and this indicates that the rarefying process has not spread deep into the bone around the tooth.

In the final stage of the disease the teeth fall out owing to the extent of bone destruction. The specimen shown in fig. 14 is an excellent example. The premolars and molars have fallen out and the alveolar process has entirely disappeared. The condition of the mandibular canine shows how the final loss of the tooth is brought about. In the living subject this advanced stage of the disease is denoted by the absence of "ridges."

FIG. 14.—Final stage of the disease. The teeth have fallen out owing to the extensive destruction of the surrounding bone.

In specimens showing marked attrition of the teeth the outer aspect of the alveolar process is often thickened and the rarefaction of the bone is limited to the alveolar margins. A case of this type is shown in fig. 15. The skiagrams (fig. 16) show that the rarefying process is almost entirely limited to the surface, and has not spread deeply into the sockets. Compare the skiagraph appearance of the bone around the incisors in this specimen and in the specimen shown in fig. 10. The marked attrition of the teeth indicates that the function of mastication has been efficiently performed, and the bone around the teeth

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having therefore been kept well nourished with a plentiful flow of blood was in a favourable condition to react to injury.

A specimen in which there is greater thickening of the bone is shown in fig. 17. The position of the calculus in relation to

FIG. 15.—A specimen in which the bone shows marked reaction. Note the thickened margin of the alveolar process.

FIG. 16.—These skiagrams of fig. 15 show that the rarefaction of bone is to a great extent limited to the surface.

the margin of the bone is evidence that well-defined pockets existed around the teeth. The surface of the bone around the tooth shows signs of a rarefying osteitis, but there is no cup-like

absorption of the bone. The thickened bone beyond the surface indicates a "sclerosing" inflammation. The arch is well formed and the attrition of the teeth on the masticating surface points to good functional activity.

An example of the formation of nodular masses of bone is shown in fig. 18.

In practice a type of case is met with in which the bone destruction on the labial and palatal aspects of the teeth is much more advanced than on the approximal aspect. In many of

FIG. 17.—A specimen from a very chronic case showing considerable thickening of the margin of the bone.

these cases the gum margin is very little above the neck of the tooth, and a probe inserted between the gum and tooth will pass almost to the end of the root. This condition is generally associated with the anterior teeth, and not infrequently with patients whose teeth are unduly prominent. A specimen illustrating the morbid anatomy of this type of case is shown in fig. 19. The bone on the labial aspects of the right canine and central incisor has completely disappeared; the left central incisor and canine are only covered by bone in the neighbourhood of the apex. Note the deposit of calculus around the necks of the teeth and the freedom from calculus of the remaining exposed portion of the roots. On examining the position of the

teeth in relation to the bone, it will be seen that the arch is decidedly narrow and that the general direction of the teeth is too vertical.

FIG. 18.—A specimen showing the formation of nodular masses on the outside of the alveolar process.

FIG. 19.—In this specimen the bone destruction is most marked on the anterior aspects of the teeth.

A point worthy of attention is that the disease spreads in the maxilla more actively and extensively than in the mandible; especially is this the case in the region of the molars. The

more limited area of bone destruction in the mandible is due to the nature of the osseous tissues, the bone in the maxilla being of a much more cancellous character than in the mandible.

FIG. 20.—Mandible from an advanced case of periodontal disease in the region of the mandibular molars.

The right premolars and molars.

The left premolars and molars.

FIG. 21.—Skiagrams of the specimen shown in fig. 20.

This difference in the spread of the disease in bones of unequal density is more fully dealt with in the chapter treating of the disease in the lower animals.

A mandible showing extensive destruction of the bone in the region of the molars is shown in fig. 20, and the skiagram of the specimen in fig. 21.

The series of specimens just described indicates that the bone lesion is a progressive rarefying osteitis which commences at the margin of the tooth socket and eventually destroys the bone until the tooth is shed owing to the loss of its attachments.

FIG. 22.—Teeth from a case associated with multiple arthritis.

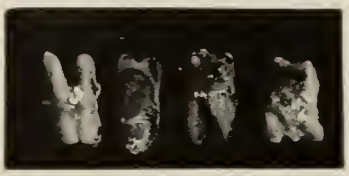


FIG. 23.—Teeth from an advanced case which had been treated for two years with vaccines.

THE TEETH.

The teeth from cases of periodontal disease exhibit definite changes. The hard tissue shows absorption, and the periodontal membrane is thickened—indications of a chronic inflammatory process. Teeth removed from a series of cases are shown in figs. 22, 23 and 24. Those depicted in fig. 22 were taken from a man, aged about 34, who was a mouth-breather and was suffering from mucous colitis. A year previously he had had an attack of arthritis involving several of the large joints.

When the hard tissues around the apex of a tooth are being absorbed the presence of periodontitis is indicated, and periodontitis necessarily implies pathological changes in the adjacent bony tissue. The degree of rarefying osteitis may be gauged

by the rapidity of tooth absorption and the liability to direct infection of the tissues increases in proportion to the amount of rarefying osteitis. The condition of the teeth in fig. 22 indicates that there was marked rarefying osteitis and probably direct absorption of toxins, &c., into the blood-stream.

Large masses of adventitious tissue are frequently found in the cleft between the roots of the molar teeth, indicating extensive loss of osseous tissue at those points. The condition of the teeth in cases exhibiting deep pockets and profuse suppuration is well shown in the specimen illustrated in fig. 23.

FIG. 24.—Teeth showing changes at their apices.

In many cases of periodontal disease the teeth become very brittle. There is some doubt as to the real cause of this brittleness. Mr. J. G. Turner attributes it to malnutrition of the dentine caused by toxins absorbed via the cementum. My own view is that the fragility is due to increased calcification of the soft parts of the dentine and cementum, and that the process is as follows: The toxins injure the tooth tissues, which, like other tissues of the body, react, the reaction taking the form of an increased calcification of the soft contents of the dentinal tubes and cemental lacunæ, thus causing greater fragility of the tooth, and rendering the tooth more brittle.

I have arrived at this conclusion after staining in borax carmine a series of teeth from cases of periodontal disease and a careful examination of the teeth themselves. On being held

to the light such teeth appear more translucent than normal teeth, especially about the apices, and if the teeth are immersed for six or eight weeks in borax carmine it will be found that the translucent area does not stain to the same extent as the other parts of the teeth, which indicates that the soft tissue normally present has undergone calcification.

Sections of teeth from three cases of periodontal disease are shown in figs. 25 to 27.

FIG. 25.

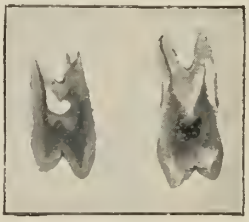


FIG. 26.

Teeth from cases of periodontal disease showing translucent areas.

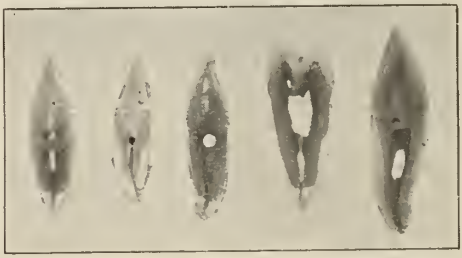


FIG. 27.—Teeth from a case of periodontal disease showing translucent areas.

PERIODONTAL DISEASE IN ANIMALS.

Periodontal disease frequently occurs in horses, in cats and dogs and captive wild animals, and it is more rarely met with in animals in the wild state. A study of the disease in these animals is very instructive, and throws much light on the etiology and pathology of this serious affection. It will be convenient to consider this section under two headings: (*a*) wild animals and (*b*) domestic animals.

(*a*) *Wild Animals.*

The Odontological Section in the Museum of the Royal College of Surgeons of England contains a unique collection of skulls, showing the disease in a wide range of animals, including rodents, marsupials, herbivora, monkeys, edentata, and the small and large carnivora.

FIG. 28.—*Macropus bennetti* (Bennett's wallaby), showing an early stage of periodontal disease. The loss of bone is more marked in the maxilla than in the mandible.

FIG. 29.—*Petrogale penicellata* (brush-tailed kangaroo). In this specimen the disease is more advanced than in specimen fig. 28.

Four specimens in this collection showing various stages of the disease in the kangaroo will be considered first.

(1) A Bennett's wallaby (*Macropus bennetti*, fig. 28) exhibits an early stage of the disease; the septa between the anterior

teeth of the maxilla have been partially destroyed, while in the region of the maxillary first molars the bone shows signs of rarefying osteitis.

(2) The skull of a brush-tailed kangaroo (*Petrogale penicellata*, fig. 29) shows a further stage, the disease being more generally distributed and the mandibular teeth more affected than in specimen fig. 28; a slight deposit of calculus is seen on the teeth.

FIG. 30.—*Macropus*. Species unnamed. In this specimen the disease is well advanced, more especially in the region of the maxillary molars.

FIG. 31.—*Macropus*. Species unnamed. The majority of the teeth have been lost from the disease.

(3) In the skull of a macropus (species unnamed, fig. 30) the disease is still further advanced, the bone being extensively destroyed in the neighbourhood of the right maxillary third molar; signs of rarefying osteitis are plentiful, while in the

mandible there is distinct thickening of the bone around the right first molar.

(4) In the left half of the skull and mandible of a kangaroo all the molars have been lost except one in the maxilla and one in the mandible, and around these teeth the bone has been extensively destroyed (fig. 31).

In the carnivora a similar gradational series can be traced, the point of special interest being that the disease tends to spread in the maxilla more rapidly than in the mandible, owing probably to the former bone being less dense in character. This difference in the spread of the disease is well shown in the specimen of a common wolf (*Canis lupus*) depicted in fig. 32.

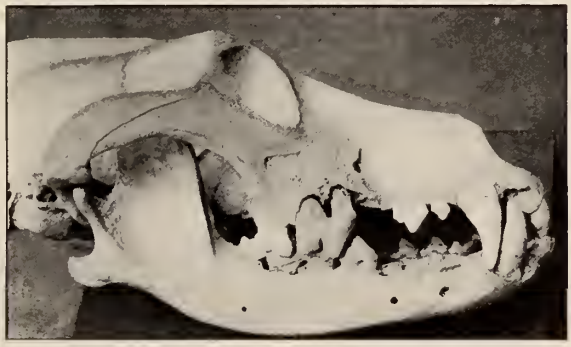


FIG. 32.—*Canis lupus* (wolf). The disease has spread more in the maxilla than in the mandible.

The bone around the maxillary fourth premolar has almost disappeared. The cup-like absorption of the socket can be well seen and the large area of rarefying osteitis indicates the spread of infection into the bone. In the mandible the disease is not so advanced and the outer layer of bone does not show signs of rarefying osteitis.

Among the suricates the mandible is more often affected than the maxilla, and this may be due to the shape and arrangement of the teeth of this animal. The mandibular molars are often slightly crowded and the shape of the teeth is such as to assist the lodgment of food. The mandible of one of these animals is depicted in fig. 33. It will be observed that the

setting of the premolars and molars is slightly irregular, that the alveolar process has been extensively destroyed, especially in the region of the molars, and that the bone absorption is greater between the teeth than on the buccal and lingual aspects.

FIG. 33.—*Suricata tetradactyla* (suricate). Mandible showing the slightly irregular arrangement of the molars and premolars.

FIG. 34.—*Cervs eldii* (Panolia deer). The destruction of the bone is most marked in the region of the first molars.

In the herbivora the disease is common; but rarefying osteitis of the outer alveolar plate is comparatively rare, and this is a point of special significance in view of the fact that, as compared with the carnivora, the bone of the maxilla in the herbivora is more dense and there is a smaller proportion of

cancellous bone. It seems probable, therefore, that the rapidity in the spread of the disease is to some extent governed by the density of the bone. A specimen illustrating the resistance of the bone in the herbivora is seen in the skull of a Panolia deer, *Cervus eldii*, shown in fig. 34.

Another point of interest in this skull is that the destruction of tissue is greatest in the region which bears the main force of mastication. In the recent state the spaces formed between the teeth are choked up with fodder.

A more advanced stage of the disease is seen in a Schomburgk's deer (*Cervus schomburgki*) (fig. 35).

FIG. 35.—*Cervus schomburgki* (Schomburgk's deer). An advanced stage of the disease.

This specimen serves to emphasize the limitation of the infection of the bone in the herbivora. Although the disease was present in an aggravated form, the bone was only very slightly affected beyond the immediate neighbourhood of the teeth.

In the specimen shown in fig. 36 the infection around the mandibular teeth has spread to the body of the bone and has resulted in extensive necrosis of the coronoid process.

This tendency to excessive suppuration in the body of the bone may be due to the shape of the teeth in herbivora. The teeth are of the hypsodont type, and the infection from

the pockets around the teeth is carried via the periodontal membrane deep into the substance of the bone and spreads rapidly in the cancellous tissue, the spread to the coronoid process and not to the condyle being again accounted for by the former offering a path of least resistance.

FIG. 36.—Cervus (species unnamed). In this specimen there has been suppuration in the body of the mandible from infection around the teeth.

FIG. 37.—*Ursus ornatus* (spectacled bear). The disease in this specimen probably followed on injury to the right maxillary canine.

The theory that the disease may be started by an injury is supported by such cases as that shown in fig. 37. The skull is that of a spectacled bear (*Ursus ornatus*) from the Andes of Peru. This animal was in the Zoological Society's Gardens for nearly fourteen years, and at its death the right side of the mouth

presented the condition shown in fig. 37. The teeth that remain are only slightly fixed to the bone and are heavily coated with salivary calculus, a large mass being adherent to the buccal aspect of the maxillary right molar. The teeth on the left side show an early stage of the disease. The interesting feature of this specimen is that the pulp of the right maxillary canine had died owing to the exposure of the pulp chamber through attrition or fracture, and it is highly probable that the trouble to the canine accounted for the severity of the disease on the right side by creating a tender area, and so interfering with the functional activity of the teeth on that side of the mouth.

FIG. 38.—*Canis familiaris* (pug dog). The disease is limited to the mandibular incisors, which are functionless teeth.

Animals in the wild state are by no means immune to pathological changes in the alveolar process, and in a recent examination I have made of the collection of skulls of monkeys in the British Museum several examples of definite periodontal disease were noticed. The feature of interest was that animals in certain localities seemed to be especially liable to the disease.

(b) Domestic Animals.

Among domestic animals the cat, the dog, and the horse are the chief sufferers from periodontal disease.

From the dogs we are able to obtain some instructive data concerning the disease. In the short-muzzled breeds, such as

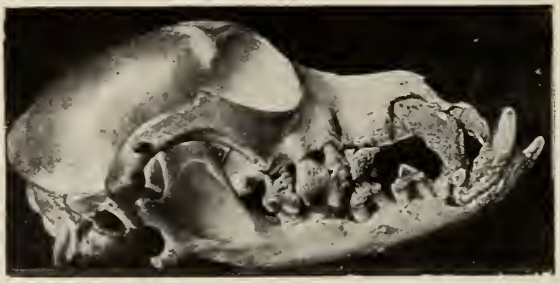


FIG. 39.—*Canis familiaris* (variety unnamed). An advanced stage. The disease commenced around the mandibular incisors.

FIG. 40.—*Canis familiaris* (Maltese terrier). An advanced stage of periodontal disease in a "Society pet."

the pug and the bull-dog, the disease usually starts around the mandibular incisors, the maxillary incisors becoming subsequently involved (fig. 38).

Towards the back of the mouth a common starting-point of the disease is the region of the second and third premolars. In

the short-muzzled breeds these teeth are often placed obliquely to the line of the arch, and are so crowded as to form excellent crevices for the lodgment of food. In the long-muzzled breeds the disease usually starts between the premolar and molars, a frequent site being the angle formed between the last premolar and the first molar on the palatine aspect. From these early beginnings the disease can be traced through all stages to the final loss of the teeth through complete destruction of the alveolar process as shown in figs. 39 and 40.

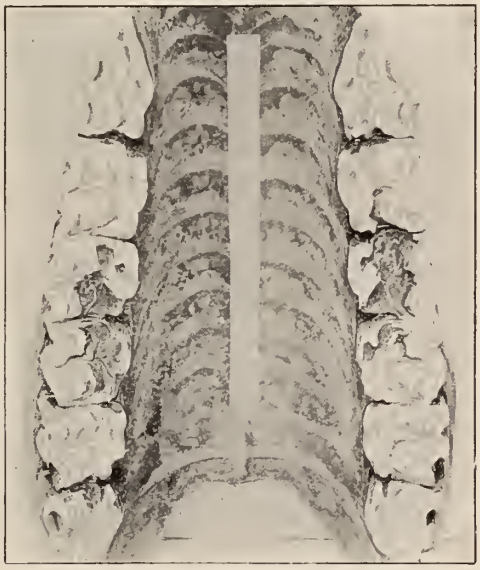


FIG. 41.—*Equus caballus* (horse). A specimen with the muco-periosteum of the palate in place. The teeth are carious. The first stage of periodontal disease is shown in the slight destruction of the muco-periosteum between the last two molars on the right-hand side of the illustration.

Among the lower animals the horse supplies us with the most instructive lessons concerning the disease, since it is possible to follow without difficulty every stage from the initial lesion to the advanced suppurative condition.

The following illustrations show the various stages of the disease in the horse.

The earliest stage is shown in fig. 41. This specimen of the

maxillæ of a horse affected with caries has been prepared with the muco-periosteum in position. The initial lesions of the muco-periosteum are seen between the last two molars on the right side of the picture and the second and third premolars on the left side.

FIG. 42.—*Equus caballus* (horse). An early stage of the bone destruction.

FIG. 43.—*Equus caballus* (horse). The bone destruction has advanced sufficiently to loosen the attachment of the first molar.

The next stage is shown in fig. 42. The loss of bone on the inner aspect of the teeth is well marked, the interproximal spaces being most affected.

A further development is illustrated in fig. 43. The bone destruction has progressed to such an extent that the first molar is quite loose and is being extruded from its socket. Here,

as in the preceding specimen, the interproximal spaces are most affected.

The formation of spaces between the teeth is shown in fig. 44, and fig. 45 gives a good idea of the condition of many

FIG. 44.—*Equus caballus* (horse). Showing spaces formed between the teeth.

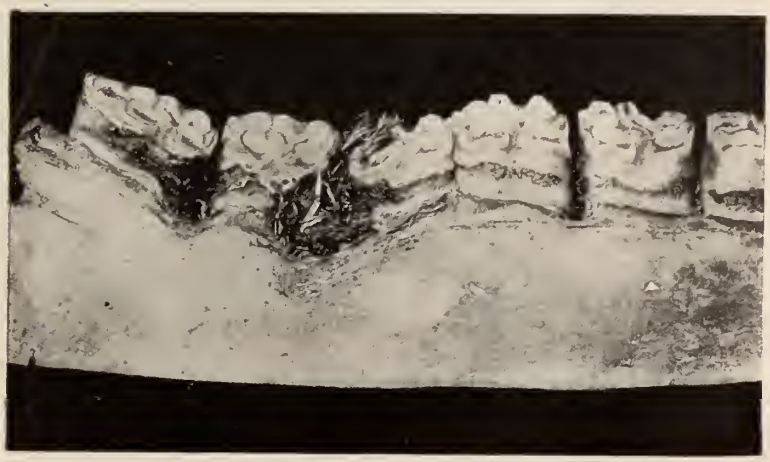


FIG. 45.—*Equus caballus* (horse). Shows the spaces filled with fodder.

of these spaces when seen in the fresh, moist condition. They are packed full with fodder and other material.

The effect of the food packing on the soft tissues is seen in fig. 46, which shows a portion of a mandible prepared with part of the muco-periosteum *in situ*. The infection from the

space between the fourth premolar and the first molar has spread to the body of the bone and has led to the formation of an abscess.

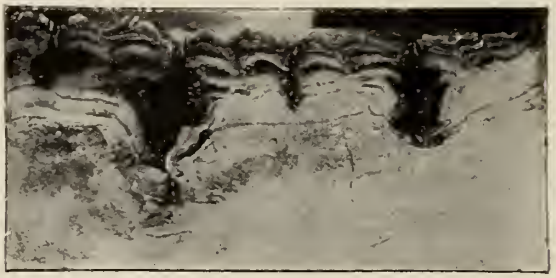


FIG. 46.—*Equus caballus* (horse). Showing the condition of the soft tissues from the food packing.

FIG. 47.—*Equus caballus* (horse). From a well-marked case of periodontal disease in which there has been suppuration in the body of the bone.

Another preparation illustrating the infection of the body of the bone from the spaces between the teeth is shown in fig. 47. The spaces are filled with fodder ; there has been suppuration in the body of the bone and considerable thickening of the outer layer of the bone.

Finally, the specimens in figs. 48 and 49 show the advanced stage in an animal which I examined when alive. Pus was flowing freely from the mouth, a fœtid discharge was flowing

FIG. 48.—*Equu scaballus* (horse). From an advanced case. A director is placed in a sinus leading to the antrum.

FIG. 49.—*Equus caballus* (horse). The left maxilla and mandible from the same case as shown in fig. 48.

from the nostrils, and the sulcus between the cheeks and teeth was clogged with food. The animal was killed and the head prepared partly as a moist specimen and partly as a dry specimen.

Fig. 48 shows the right maxilla. The premolars and molars are carious. The margin of the gum is thickened and detached from the bone. Between the second and third molars there is a huge space which communicates with the antrum, the lining membrane of this cavity being thickened and inflamed. The left half of the skull and the mandible (shown in fig. 49) give a good idea of the bone lesions. Several of the teeth, both in the maxilla and mandible, are carious, the alveolar process has been extensively absorbed, the greatest loss of tissue being in the region of the two last maxillary molars. The space between these teeth communicates with the antrum. The buccal aspects of the posterior teeth show signs of caries, which was no doubt due to the lodgment of food in the buccal sulcus. Had we not been able to observe the disease in the horse through the progressive stages shown in figs. 41 to 47 it would have been difficult to understand how such an extreme condition as is manifest in this case could have been the direct result of a slight injury to the muco-periosteum.

The conclusions to be drawn from an examination of the disease in man and other animals are :—

(1) That the bone lesion is a progressive rarefying osteitis commencing at the margin of the alveolar process, and (2) that the varying density of the bone influences the rate of destruction.

CHAPTER II.

The Microscopical Anatomy of Periodontal Disease.

THE knowledge which we possess of the microscopical anatomy of periodontal disease is based mainly on the work of Znamensky and Talbot. Dr. Znamensky has given us in detail[1] the results of his examination of sections from the jaws of a female, aged 39, who died of acute anæmia resulting from hæmorrhage after parturition. "The decalcification of the preparations was accomplished by means of a 3·5 per cent. solution of trichloracetic acid." Some of the sections were stained with eosin and hæmatoxylin and others with Hoeffler's blue. The sections show that the earliest pathological changes commence at the gum margin and that subsequently the periodontal membrane and adjacent bone are successively involved. The illustrations given in Dr. Znamensky's paper show clearly the different stages of the disease. Fig. 50 shows the earliest stage. The epithelial covering of the gum is still intact, but in the papillary layer there is an infiltration of the tissues with leucocytes. The remaining portion of the gum and the bone are normal.

The next section (fig. 51) depicts a further stage; the destruction of the epithelial layer has commenced and the gum is infiltrated with leucocytes almost to the edge of the bone.

Fig. 52, to quote Znamensky, "represents the period of the disease when the infiltration has already reached the bone, and from this moment the changes in the bone of the socket begin. First they appear upon its free rim. "For better evidence

[1] Transactions International Medical Congress, 1913 (Stomatological Section).

I took that part of the socket where, examined by the naked eye, it has the thickness of a sheet of paper, and under the microscope appears to be a narrow, long plate not containing any bone-marrow. The destructive process is proceeding in it in this manner: First the socket loses lime salts, and gets trans-

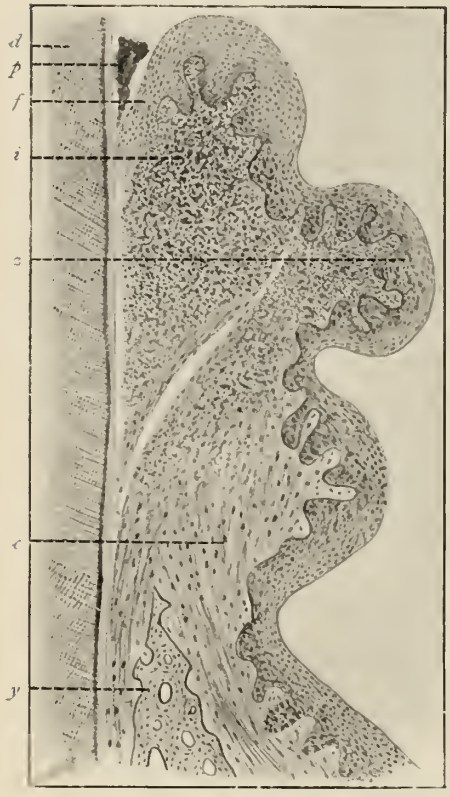


FIG. 50.[1]—*d*, dentine; *p*, calculus; *f*, papillary layer of the gum; *z*, epithelial layer; *i*, tissue infiltrated with leucocytes; *c*, normal gum; *y*, normal bone. Magnified 80 times.

FIG. 51.—A more advanced stage than that shown in fig. 50. The destruction of the epithelium has commenced at *h* and *y*. Magnified 80 times.

formed into an osteoid tissue, and afterwards into a fibrous connective tissue. Directly this is done the infiltration with white blood corpuscles begins. Those places of the thin socket which

[1] The description of the sections are copied from Dr. Znamensky's paper.

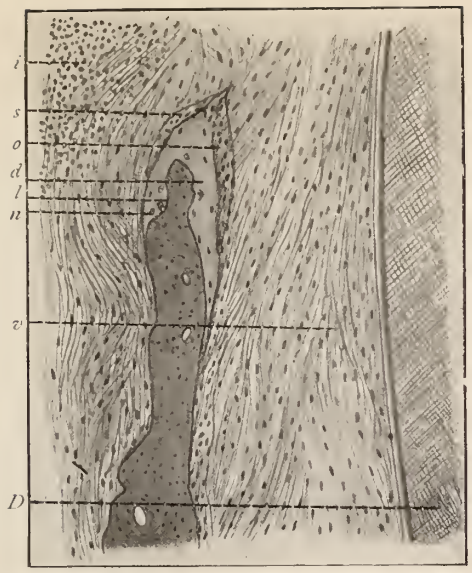


FIG. 52.—*i*, infiltration proceeding from the gum to the bone; *s*, the part next to the infiltrated part of the socket is transformed into a fibrous connective tissue; *o*, osteoid tissue; *d*, part of the bone beginning to lose its lime salts; *n*, bone corpuscles; *l*, broken line dividing the healthy part from the affected bone; *v*, alveolar periosteum not yet inflamed; *D*, dentine. Magnified 180 times.

FIG. 53.—*o*, place where the osteoid tissue has had time to be gnawed by intervening tissue, infiltrated with leucocytes; *m*, place where the bony plate of the socket has had time to be half gnawed by osteoid tissue; *i*, infiltration of the intervening tissue is more sharply expressed; *v*, periosteum of the socket beginning to react; the blood-vessels becoming wider at *k*. Magnified 180 times.

began to lose lime, when magnified by the aid of a microscope, present a uniform homogeneous semi-lucid mass, in which no bony laminæ are to be seen; and the bone corpuscles lessened in their number have lost their characteristic forms, and have become more and more like the cells of a connective tissue. The bone which has been altered in that manner is very

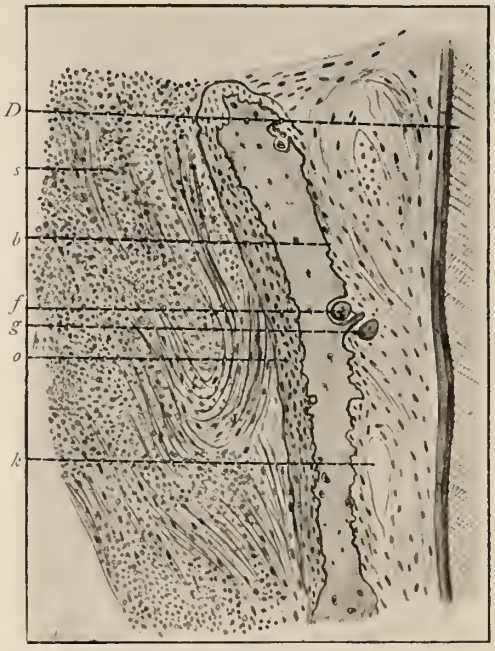


FIG. 54.—*o*, outside of the tooth socket being connected with osteoid tissue; *b*, inside of the tooth socket showing Howship's lacunæ; *g*, osteoclasts; *f*, the so-called lacunar absorption of the bone is proceeding; the blood-vessels of the periosteum, *k*, are dilated, but the periosteum is but little infiltrated, the gum, on the contrary, showing considerable infiltration; *s*, reaching the bone of the socket; *D*, dentine. Magnified 180 times.

distinctly divided with a broken line from the neighbouring healthy part of the bone, and by a similar broken line it is again divided from that other part of the bone which has already had time to change into a fibrous tissue, and the peripheral part of this latter passes quite imperceptibly into the fibrous connective tissue of the gum. Thus, microscopically examined, we see the following: beginning from the free edge,

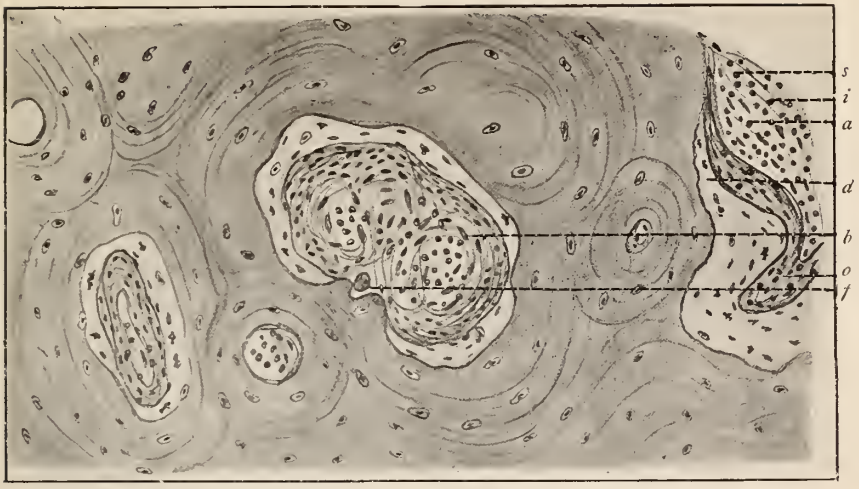


FIG. 55.—This section shows the primary alterations in the marrow part of the bone of the socket. The absorption of the bone from the side of the periosteum is proceeding, *a;* and from the Haversian canals, *b*, upon the margins of healthy bone, *i.e.*, the part which begins to get decalcified, *d*, all round the Haversian canal the inner layer of the decalcified bone is transformed into an osteoid tissue, *o*, and this latter nearer to translucent part of the Haversian canal has already passed into a fibrous connective tissue, *s*, impregnated with infiltration, *i;* the same order of alterations in bone proceeds also from the side of the periosteum of the root; *f*, osteoclasts. Magnified 360 times.

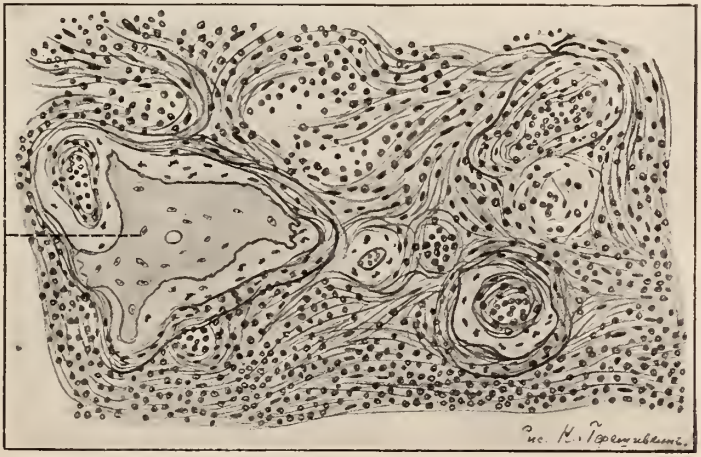
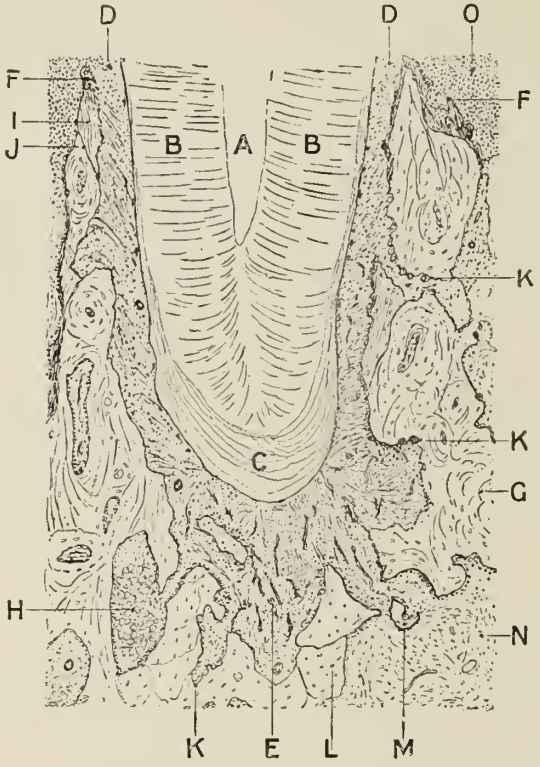


FIG. 56.—This section shows the concluding stage of the development of the disease in the marrow part of the bone; bony aminæ are absorbed and transformed into fibrous connective tissues very strongly impregnated with inflammatory infiltration. There is a sequestrum to be seen which is beginning to be absorbed from the periphery. Magnified 360 times.

a part of the bone of the socket is already transformed into connective tissue, which passes deeper into an osteoid tissue. There is a place to be found where the osteoid tissue has already



(From a drawing by Mr. A. Hopewell-Smith).

FIG. 57.[1]—A, pulp cavity; B, dentine of tooth; C, hyperplasic cementum around apex of root: D, periodontal membrane, greatly thickened—hyperplasic; E, indifferent tissue at apical region greatly increased in amount; F, free edge of bone of socket becoming converted into fibrous intervening tissue; G, bone of socket presenting earliest signs of osteoporosis; H, large osteoporotic space in bone of jaw filled with bone-marrow; I, bone of socket partially decalcified and converted into osteoid tissue; J, junction of living with decalcified bone; K, osteoclasts producing lacunar absorption; L, bone of jaw only slightly altered by disease; M, sequestrum undergoing peripheral absorption; N, soft, cancellous tissue slightly changed from normal; O, inflammation of gum at neck of tooth.

had time, so to speak, to gnaw away the bony plate of the socket across its breadth (fig. 53). At this period inflammatory reaction can be traced in the periosteum, the blood-vessels

[1] From the *Lancet*.

dilate and the tissues become infiltrated with leucocytes." A further stage of the bone destruction is shown in figs. 53 and 54.

The bone is first decalcified, then gradually transformed into an osteoid tissue and afterwards into a connective fibrous tissue which becomes infiltrated with leucocytes. In the deeper parts of the tooth socket the tissue changes are in part similar to those thus described; the so-called lacunar absorption of the bone is, however, more pronounced, the absorption proceeding not only from the periosteal surface but also from the Haversian canals. The stages of the bone destruction in the medullary part of the bone are shown in figs. 55 and 56.

The main features of the condition of the bone in advanced cases are shown in fig. 57.

Znamensky remarks that the bone changes are identical with those occurring in other diseases, for example, osteomalacia, rickets, tabes dorsalis, atrophy of old age, &c.

Talbot in his excellent work on "Interstitial Gingivitis" gives a series of illustrations depicting the microscopical anatomy of periodontal disease in dogs. He shows that the earliest manifestation of the disease is in the gingival margin, and that, as the disease progresses, both the periodontal membrane and the bone become involved. Talbot also gives illustrations of the disease in man, and his researches, which confirm the work of Znamensky, show that the changes in the bone are of the character of a rarefaction or osteoporosis which commences at the margin of the tooth sockets. The microscopical investigation of the disease thus confirms the conclusions arrived at by those who have investigated the macroscopical anatomy of the disease in man and other animals.

CHAPTER III.

The Clinical Appearances of Periodontal Disease.

THE gums normally fit close around the necks of the teeth, the spaces between the teeth being filled with tags of gum—the interdental papillæ.

The earliest indication of the disease is a slight congestion of the interdental papillæ, and even at this early stage the gum attachment will be found to be partially destroyed, as may be proved by passing a probe into the interproximal spaces.

As the disease progresses the whole of the gingival margins become congested and bleed readily; the interdental papillæ disappear and the normal festooned arrangement of the gum around the tooth is lost. An examination of the teeth at this stage will usually disclose food and other débris in the approximal spaces, while small nodules of calculus are generally present on the teeth. Compression of the gums will cause a small quantity of discharge to escape at the sides of the teeth. Eventually the bone becomes involved in the inflammatory process and is gradually destroyed together with the periodontal membrane. The gums recede, but the recession does not usually proceed so rapidly as the destruction of the alveolar process, and the result is that, around the teeth, deep pockets are formed, in which pus and other morbid material accumulate and aggravate the condition.

This stage is characterized by the following condition : The mucous membrane appears deeply congested; the gums are swollen at their free margins and bleed readily on the slightest touch; the roots of the teeth are more or less exposed, and covered with a layer of hard greenish-brown calculus; there is a fœtid discharge and the breath has a repulsive odour due to indol-forming organisms. A sickly, sour odour of the breath

is noticeable when yeasts and bacteria causing carbohydrate fermentation are present. The teeth are freely movable and may become so loose that they can be removed quite easily. If the disease is left untreated the teeth are lost one by one, and with the loss of the teeth the inflammatory process completely disappears.

The disease usually progresses with varying rapidity, and while the inflammation at one period may be acute with a free flow of pus, at a later period its progress may be slow. The activity of the disease varies around different teeth ; in some cases the greater activity is around the posterior teeth and in others around the incisors.

In some cases the margin of the gum is thickened and in a few instances well-marked bosses of bone are present on the outer aspect of the alveolar process. To a great extent the clinical appearances of the disease vary according to the general condition of the patient, the resistance of the tissues, and the hygiene of the mouth.

The disease, especially in advanced cases, is usually associated with congestion of the tonsils and the mucous membrane of the mouth and pharynx. Superficial glossitis is often present. The patient experiences an unpleasant taste in the morning. There is frequently recurring hæmorrhage from the gums, which is swallowed during the day, but occasionally escapes from the mouth at night and stains the pillow. The last symptom deserves special attention, as there is some danger that it may be mistaken for hæmorrhage from the lungs.

Careful attention to the hygiene of the mouth will not only prevent the spread of gingivitis but will also prevent the deposit of calculus around the necks of the teeth, as the formation of stagnation areas at the gingival margin is favourable to the deposit of calculus.

Mouth-breathers are specially liable to gingivitis, but if the tissues react strongly the margin of the alveolar process is thickened and the pockets around the teeth as a rule are not deep. The type of the organism present naturally influences the type of toxins produced in the pockets and so affects the rapidity of destruction of the tissues.

It is not safe to judge the extent of the disease from clinical appearances only, and it is necessary to call in the aid of

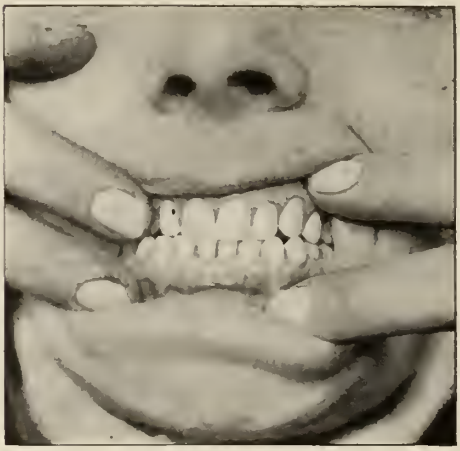


Fig. 58.

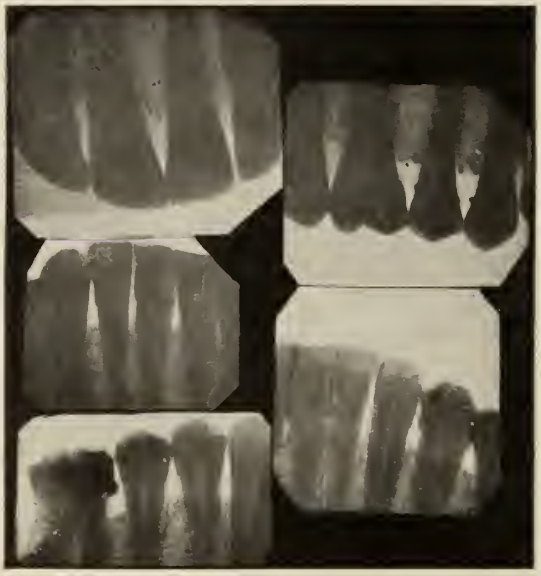


Fig. 59.

skiagraphy in order to ascertain how far bone destruction has proceeded. This is illustrated in the following cases.

The case shown in fig. 58 depicts an early stage of the disease.

Clinically there was no recession of the gums beyond a partial disappearance of the interdental papillæ between the lower incisors. There was a marked marginal gingivitis together with

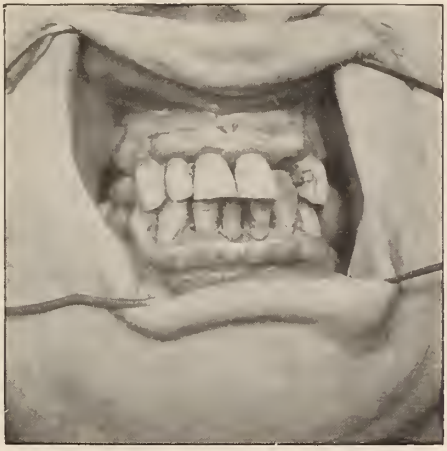


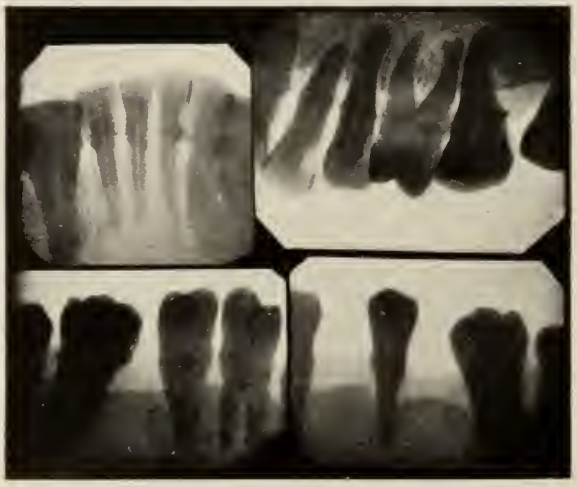
FIG. 60.

FIG. 61.

a congestion of the whole of the gums and a free discharge from the pockets around the teeth. The arch was well developed and there was marked attrition.

The skiagrams (fig. 59) show that the alveolar process was involved and that the destruction of the bone had advanced to a considerable extent in the lower incisor region.

FIG. 62.



(*a*) (*b*)

FIG. 63.

The clinical appearances in the case shown in fig. 60 suggest a more advanced condition of the disease than in the preceding case. The muco-periosteum is more swollen and congested.

The interdental papillæ have disappeared and the gum margin on the labial aspects of the teeth just fails to cover the necks of the teeth. The skiagrams (fig. 61) show that the bone destruction is much more advanced than the gum recession would seem to indicate.

The case shown in fig. 62 illustrates an advanced condition. The skiagrams (fig. 63) show the extensive destruction of the tooth sockets. Those marked (*a*) and (*b*) also illustrate a point, namely, that the calculus extends only very slightly below the gum margins.

FIG. 64.

The case shown in fig. 64 illustrates some features which are of interest from the point of view of prognosis. The patient, a man, aged 34, was a compositor by trade, and attended the out-patient department for dyspepsia.

Beyond a well-defined marginal gingivitis the gums were normal in colour and firm in consistency. The teeth were smothered with calculus, the gum margin was above the necks of the teeth, and the interdental papillæ had disappeared. Examination with a probe showed slight pockets around the teeth.

From the clinical appearance it would be inferred that the disease was of slow progression and that there was comparatively little bone destruction.

The skiagrams (fig. 65) show that the bone destruction is

proceeding with the same rapidity in all parts of the mouth, and that the inflammatory process extends only slightly beyond the margin of the bone.

FIG. 65.

FIG. 66.

In the case shown in fig. 66 the patient was a mouth-breather and complained of bleeding from the region of the mandibular incisors. The teeth had been regularly cleaned. The gums were well up to the level of the necks of the teeth and the interdental papillæ were not destroyed ; there was no gingi-

vitis except around the mandibular incisors where pus was present. An examination with a probe showed extensive destruc-

FIG. 67.

FIG. 68.

tion of the alveolar process in the region of the mandibular incisors, while in the rest of the mouth the bone was intact.

The skiagrams shown in figs. 67 and 68 illustrate these points.

CHAPTER IV.

The Pathology of Periodontal Disease.

Considerable light has been thrown on the pathology of periodontal disease by studying the disease in animals. The study of a certain disease in the animal kingdom will often assist us in arriving at a truer conception of the same disease when attacking man. This is certainly true of periodontal disease, and it will therefore be an advantage, before discussing the problem as affecting man, to pass in review the facts we know regarding the pathology of the disease in the lower animals.

(1) Horses. — In the earlier description of the morbid anatomy of the disease in the horse the initial lesion was shown to be a slight destruction of the interdental papillæ probably from the injurious character of the food. The injury is usually found on the buccal side in the maxilla and on the lingual side in the mandible. Food and other matter soon accumulate at the injured spot and stagnation areas are formed. Organisms already present or freshly introduced into the mouth add infection and the toxins produced cause further destruction of tissue. The stagnation areas are thus increased, and the tissue destruction is accelerated. The infection tends to spread, in the maxilla, to the antrum, and, in the mandible, to the body of the bone leading in both cases to suppuration.

In the stagnation areas the teeth become painful to pressure and the function of mastication is naturally transferred to other areas, with the result that the accumulation of food débris around the affected teeth is promoted.

In the horse the disease is local in origin and is primarily due to traumatic injury. Subsequently the lesion spreads partly

from trauma arising from the tightly wedged accumulation of food débris and partly from toxins produced in the stagnant areas.

(2) Cats and Dogs. — Periodontal disease is exceedingly prevalent among domestic cats and dogs. Mr. H. Gray, who as a veterinary surgeon has had a wide experience of the disease in cats, is of the opinion that the highly bred pet animals which are fed on soft food are much more susceptible to attack

Fig. 69.—*Felis domestica* (cat). Appearance of normal gums.

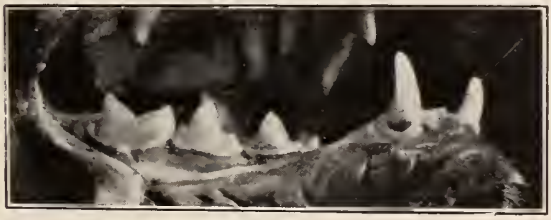


Fig. 70.—*Felis domestica* (cat). The gums show a marginal gingivitis.

than the ordinary domestic cat fed on a meat diet, the latter being comparatively free from the disease. The explanation is that the soft diet clings about the teeth, stagnation areas are formed, and a marginal gingivitis is produced. Such a condition is well shown in figs. 69 and 70.

Destruction of the tissues forming the tooth socket follows as a direct result of injury from toxins produced in the stagnant areas.

Among dogs the disease is met with most often in the pampered lap dogs and in the short-muzzled breeds of dogs,

such as the pugs and bull-dogs, and as a rule it is the non-functional teeth that are affected, namely, the incisors. In the long-muzzled types the disease very commonly starts in the region of the second and third maxillary premolars. Generally speaking, therefore, the disease commences in dogs in positions where soft food is apt to rest, and, as dogs that feed on flesh are invariably free from the disease, it is only reasonable to infer that in dogs, as in cats, the initial lesion in the gingivæ is to be attributed to the lodgment of food débris.

(3) Wild Animals.—Periodontal disease is common in wild animals kept in captivity. It is found in rodents, monkeys, marsupials, herbivores, and the carnivores. On the other hand, the disease is uncommon in animals living in the wild state.

It will be noticed that in the skulls of captive animals affected by the disease the bone destruction is usually farthest advanced around the areas where the greatest force of mastication is exerted and consequently where the soft tissues surrounding the teeth are most liable to injury. The frequency of the disease in captive animals and the comparative immunity of animals in the wild state, taken in conjunction with the limitation of the disease to the masticating area, point to the disease being closely associated with the character of the food given to captive animals. This food is generally of a softer character, needing less rending and tearing than the food which the animals would obtain in the wild state, consequently the teeth are less used and the natural cleansing operations are not performed. When captive wild animals are given food of a character similar to their natural food, as in the case of the large carnivores which are fed on a diet of fresh meat, they are almost entirely free from the disease.

Thus, from a study of the teeth of animals we are led irresistably to the conclusion that the disease originates locally through food lodgment, the tissues being damaged by the fermentation and putrefaction of the food. This view obtains additional support from the condition of the gums and teeth of cattle in parts of America where a lesion of the gums frequently occurs owing to the penetration of the tissues by the barbed

crowns of *Hordeum jubatum*, leading to destruction of the teeth sockets and loss of the teeth.[1]

Talbot[2] states that interstitial gingivitis is very common in cows fed upon brewers' slop and confined indoors. The slop diet accumulates about the teeth and directly injures the gingival margin.

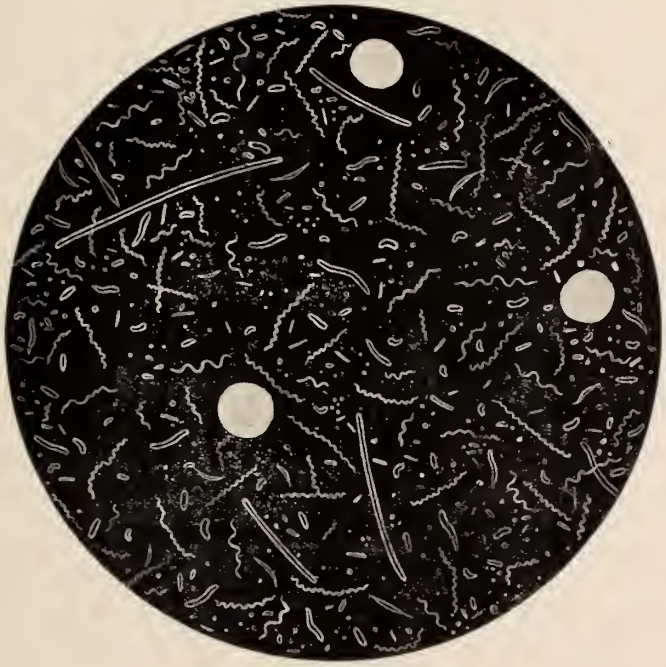


FIG. 71.

(4) MAN.—If a cover-slip preparation is made from a case of periodontal disease a very heterogeneous collection of organisms will be found. In fig. 71 such a preparation is seen, under the dark ground illumination, and Dr. Penfold, who kindly examined the slide, states that the following types of organisms are present :—

(1) Treponemata of *macrodentium* and *microdentium* varieties, so-called spirochætes of the mouth.

[1] "Dental Surgery," by Tomes and Nowell, p. 648.
[2] "Interstitial Gingivitis," p. 126.

(2) Vibrios and spirillary forms.

(3) Bacilli in great variety.

(4) Filamentous forms.

(5) Cocci of various sizes, the grouping of which cannot be determined without cultivation methods.

The large round bodies in the preparation are blood cells.

The discharge from the same pocket was examined by means of transmitted light and revealed the presence of *Entamœbæ buccalis;* a drawing of four stages of one amœba is reproduced in fig. 72.

Fig. 72.

The organisms generally present in cultures made from cases of marginal gingivitis are staphylococci, streptococci, with at times the *Micrococcus catarrhalis* and the pneumococcus.

In a series of thirty-three cases recorded by Eyre and Payne[1] the following organisms were regarded as responsible for periodontal disease :—

	Cases
Micrococcus staphylococcus pyogenes aureus	2
Micrococcus catarrhalis	9
Micrococcus catarrhalis and *Streptococcus pyogenes longus*	11
Streptococcus pyogenes longus	7
Streptococcus lanceolatus pneumoniæ (pneumococcus)	4

In a more recent communication Dr. L. S. Medalia[2] gives the results of his study of 115 cases. He found that the organism most frequently met with was the pneumococcus (*Streptococcus lanceolatus pneumoniæ*), 107 times out of 115. The results of his investigation are as follows :—

[1] *Proc. Roy. Soc. Med.* (Odont. Sect.), vol. iii, p. 36.

[2] *Dental Cosmos*, 1913, p. 24.

	Cases
Pneumococcus strepto-lanceolatus pneumoniæ	26
„ and staphylococcus	67
„ and streptococcus	3
„ staphylococcus and streptococcus	10
„ and *Micrococcus catarrhalis*	1
Staphylococcus and *Micrococcus catarrhalis*	2
Staphylococcus aureus	2
Streptococcus and staphylococcus	1
Sterile	3

From the foregoing it will be noted that the germs found in the pus from cases of periodontal disease are the same as those responsible for ordinary suppuration in other parts of the body.

Noguchi[1] has shown that in the mouth there are two definite varieties of spirochætes, the *Spirochæte microdentium* and the *S. macrodentium*, and Drs. J. G. and D. Thomson[2] have shown that in severe pyorrhœas spirochætes are usually present.

Recently Barrett and Smith, Bass and Johns, and others have called attention to the frequency of the association of amœbæ with pyorrhœa alveolaris, and it has been claimed that the cause of periodontal disease is *Endamœba buccalis*. In 1849 Gros first observed amœbæ in the mouth. Sternberg (1862) found them in the sordes about teeth.

The active forms of *A. buccalis* are from 6 μ to 32 μ in diameter; they average about 15 μ. They have a distinct ectoplasm, well seen when the organism is in motion; it is hyaline and refractile. The endoplasm is granular and shows many food vacuoles, but no contractile vacuole. The nucleus is well defined and shows a thick greenish membrane containing a great deal of chromatin. A small centriole is situated near the centre of the nucleus. The motility is sluggish and the pseudopodia are blunt. The cysts of the organism have never been seen in the mouth. The above description is accepted by the American workers.

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[1] *Journ. Exper. Med.*

[2] *Proc. Roy. Soc. Med.*, vol. vii, No. 9. Some researches on spirochætes occurring in the alimentary tract of man and some of the lower animals.

There is, however, little doubt but that the amœbæ of the mouth are of more than one species. The points in favour of the amœba being the cause of the disease are : (1) Its frequent association with the disease; (2) its power to phagocyte red cells, a power not possessed to any degree by free-living amœbæ; (3) improvement in the local conditions stated to follow the use of emetine; (4) the resemblance of the *A. buccalis* to *Entamœba histolytica*, the cause of amœbic dysentery.

The points against *A. buccalis* being the cause of the disease are: (1) The constant association with the disease of other organisms, *e.g.*, *spirochætes* or *pneumococci;* (2) emetine treatment removes the amœbæ but does not cure the condition ; (3) the disease has never been produced by inoculation of a suitable animal with a pure culture of the organism. The question is still undecided, but the pyorrhœal pocket may very well be simply a favourable environment for the growth of free-living amœbæ.

To sum up, investigations into the bacteriology of the disease do not afford any evidence of its being due to a specific organism.

With this brief survey we must leave the bacteriology of the disease and consider the condition known as marginal gingivitis, an affection which is the forerunner of periodontal disease.

The removal of all débris is brought about and the gingival margins kept in a healthy condition by—

(1) Efficient mastication of suitable foods.

(2) Friction of lips and cheeks against the gum margins.

(3) Movement of the tongue against the teeth.

(4) Free flow of currents of saliva.

Abnormal conditions, such as mouth-breathing and functionless teeth, lead to the accumulation of food débris, &c., at the gingival margin ; the material thus lodged undergoes fermentative and putrefactive changes, the soft tissue is injured, and inflammatory reaction follows. In mouth-breathers this process can be plainly seen. If the mouth of a child suffering from nasal obstruction be examined, a marginal gingivitis will be found

around the anterior maxillary and mandibular teeth, while the gingival margin at the posterior part of the mouth will be found quite healthy, provided, of course, that the function of mastication is properly performed. With mouth-breathing the natural friction of the lips against the gums is, to a great extent, in abeyance, and the result is that the débris around the teeth is not removed and the gingival margin is injured. The gingivitis so often associated with many fevers arises, partly at least, from a similar cause. The fevers frequently give rise to a condition which induces mouth-breathing, and, a "slop" diet being necessary, the ordinary functions of the mouth are not performed and food naturally collects at the gingival margins. If the marginal gingivitis persist, the periodontal membrane becomes involved and the condition known as periodontal disease supervenes.

In non-mouth-breathers the disease commences in the molar region and gradually spreads to the anterior teeth, while in mouth-breathers the disease commences earlier in life and is not infrequently confined for some time to the anterior teeth. Thus in the non-mouth-breathers the disease commences in the region where lodgment of food is liable to occur and the initial lesion is probably an injury to the gingivæ from food débris. In other words, the disease commences precisely in those positions where stagnation areas are most likely to form ; a marginal gingivitis is started and in time the periodontal membrane becomes involved, the tissue destruction following exactly the same course as in the lower animals.

The initial lesion in man is injury to the gingival margin leading to the formation of a stagnation area. From the material which collects in this area toxins are formed and the tissue changes which follow are identical with those seen in the lower animals. In the area immediately beyond the ulcerating surface —for as such it must be regarded—the tissues react and form a protecting barrier, and the progress of the disease depends on the efficiency of this barrier. The reaction of the tissues depends upon—

(1) Their inherent vitality.

(2) The degree of functional activity—the greater the flow of lymph through the area the greater will be the accumulation of antibodies.

(3) The severity of the infection.

In many cases of periodontal disease, more especially in young adults, the natural defence of the tissues suffices to prevent the undue absorption via the bone of toxins or organisms. The discharge from the mouth, however, is swallowed with the saliva and consequently the gastric mucous membrane is continually being damaged. Sooner or later the defence of the tissues at this

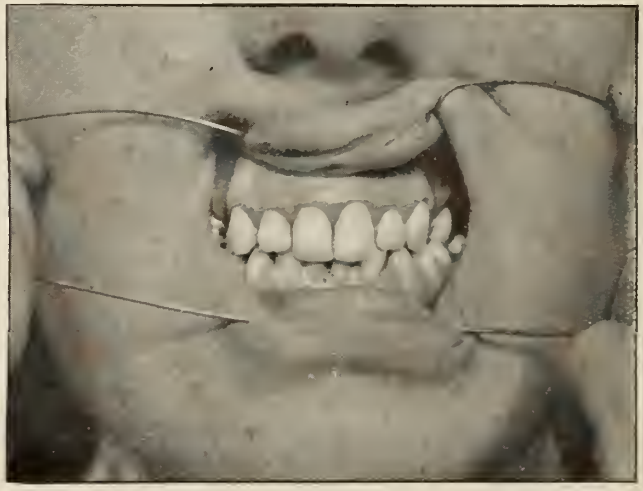

FIG. 73.

part breaks down and eventually the whole intestinal tract becomes involved.

The tissues of many patients seem to be incapable of raising a sufficiently effective barrier even in mild cases. From the pockets around the teeth a direct absorption takes place through the periodontal membrane and pathological changes commence in the tissues around the apices of the teeth. From these areas the toxins pass into the general circulation.

The view here expressed is well illustrated by the two following cases :—

M. P., single, aged 30. This patient came under observation in January, 1910. The condition of the gums is shown in fig. 73. A general gingivitis was present, but the recession of the gum margins was slight.

The pockets around the teeth were moderately deep, the gums bleeding readily on the slightest touch. The skiagrams (fig. 74) show the condition of the alveolar process in the incisor region. The patient was a pronounced mouth-breather and occasionally suffered from indigestion, but she stated that she generally felt

FIG. 74.

well. In 1904 she was assured for £100. Scaling and local remedies, combined with Bier's treatment, were tried. In July, 1910, the appearance of the gums had improved, although a good deal of gingivitis was still present. Skiagrams taken did not show any extension of bone destruction.

In October, 1910, the patient developed lung trouble, and was treated in a home, and at Easter, 1911, she had an attack of diphtheria. In June the mouth condition was worse, and the following teeth were removed :—

8 7 5	8
8 7	4 5 7 8

From the apex of $\underline{5|}$ the *Micrococcus tetragenus* was obtained. The removal of the upper and lower incisors was carried out early in September. On October 9 the patient stated that there

Fig. 75.

Fig. 76.

had been distinct improvement in her health since the last extractions, and she had gained 8 lb. in weight. Skiagrams

of the alveolar process taken immediately before the extraction of the premolars and molars are shown in fig. 75 and of the incisors in fig. 76.

During the treatment skiagrams were taken at frequent intervals, and showed very slight increase in the amount of bone destruction during the eighteen months of local treatment. The teeth removed from this patient showed definite signs of trouble round the apices, indicating the presence of a rarefying osteitis (see fig. 77).

In this case there are several points of interest. The gingivitis persisted, notwithstanding that the patient paid the most scrupulous attention to the cleanliness of her mouth, and

FIG. 77.

the lesson to be learnt from this is that no care and cleanliness on the part of the patient who is a mouth-breather will remove the gingivitis, and therefore the dental sepsis, when once it is established. The alveolar process had been only very slightly destroyed and this would seem to indicate that the Bier's treatment had proved of some value, but too much importance must not be laid on this point. Another question that presents itself is this: If the teeth had been removed early in the previous year, would the avoidance of six months of dental sepsis have prevented the development of lung trouble? Lastly, the acceptance of this patient as a first-class life raises a point of considerable interest in social and commercial circles. The

patient is suffering from an incurable condition of marked oral sepsis which is admittedly one of the most important etiological factors in the whole realm of pathology, and the question very

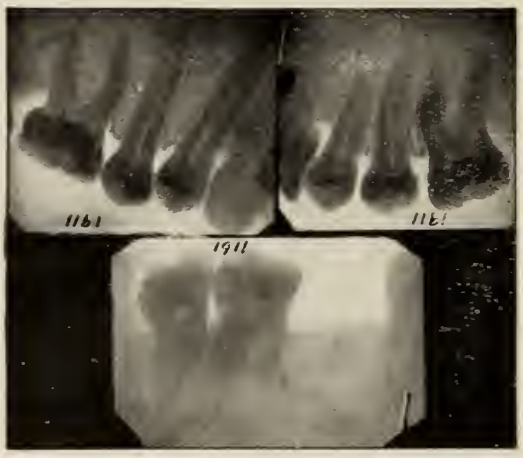

FIG. 78.

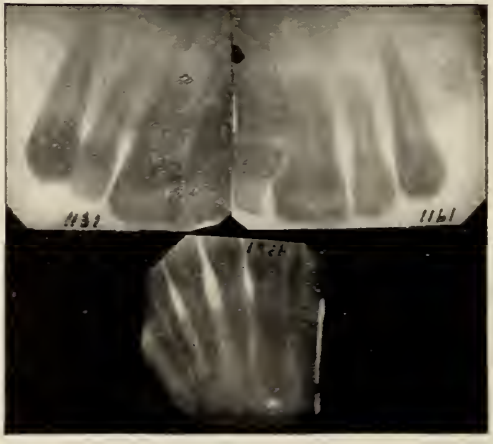

FIG. 79.

naturally arises whether she could properly be regarded as a first-class life with such a potential cause of disease present.

The second case is that of a female, aged 25. She had

marked general gingivitis and was a mouth-breather. Local treatment had been carried on since the previous October. The patient complained that she was never without headaches and that she felt "terribly shaky down to the bottom of her stomach." The slightest exertion made her feel sick. Her tongue was coated. Her blood-pressure was 141. Skiagrams of this patient are shown in figs. 78 and 79.

In a few places there were signs of rarefying osteitis. With experience of previous cases, and taking into consideration the general condition of the mouth and the health of the patient, the removal of all the teeth present was advised, namely—

$$\frac{7\ 6\quad 3\ 2\ 1\ |\ 1\ 2\ 3\ 4\quad 7\ 8}{6\ 5\ 4\ 3\ 2\ 1\ |\ 1\ 2\ 3\ 4\ 5\ 6\ 7}$$

By June 7 the teeth had been extracted and, without exception, showed pathological changes at the apex. Towards the end of July the blood-pressure had fallen to 124, the sickness had gone, the skin was clearer, and the patient admitted that she was feeling much better. By the end of October she had increased 6 lb. in weight, was feeling very much better, and eventually made a complete recovery. The above cases are merely given as typical examples of many others.

Teeth removed from cases of periodontal disease usually show a deposit of calculus; in some cases the deposit occurs as a rim of hard calculus just below the neck of the tooth, while in others the calculus is freely dispersed over the surface of the root.

Many observers attach considerable importance to the calculus around the necks of the teeth and regard this as the cause of periodontal disease. Dr. Black regards the gingival organ as a glandular structure and considers that it possesses the function of selecting and eliminating from the blood certain poisons, as evinced in mercurial gingivitis. The elimination of these poisons leads to inflammatory reaction in the gingival tissues, and, partly from the abnormal secretion and partly from the inflammatory exudates, the calculus is formed.

The presence of a stagnation area is, in my opinion, quite sufficient to account for the deposit of calculus, and I am

inclined to regard the deposit as the product of the material stagnating in the pockets. When calculus has been deposited, drainage of the pockets around the teeth is hindered and the calculus consequently increases through stagnation, and the progress of the disease is assisted. Clinical evidence supports the view that the deposit of calculus is the result and not the cause of the gingivitis.

The view that the disease is intimately associated with the deposit of urates in the periodontal membrane does not seem to be tenable in the light of facts obtained by recent observers. Dr. Pierce, who is the main advocate of this view, states that the salts are most frequently deposited near the region of the apex in the form of calcium and sodium urates with traces of uric acid and calcium phosphates. The deposit causes irritation, the tissues break down, an exit is formed for the pus around the neck of the tooth, and infection of the mouth ensues.

In connection with this contention it must be remembered that gout is essentially a non-suppurative disease and gouty deposits in the hands, ears, &c., do not promote suppuration. It is extremely doubtful whether the so-called gouty periodontitis can be regarded as a clinical entity. It is probable that the appearances seen in patients of a gouty diathesis are due to a chronic injury of the periodontal membrane of low intensity and that the adjacent tissues react and give rise to the clinical appearances classed as gouty periodontitis.

In considering the pathology of periodontal disease, careful attention must be given to the degree of resistance of the osseous tissues, which has probably a very important bearing on the rate of progress of the disease. From an examination of dried specimens it would certainly appear that the more compact the osseous tissue the greater the barrier to the spread of the disease. The question arises whether the tissues of the tooth socket are as resistant to disease as might naturally be expected. We are without any trustworthy data bearing on this point, but there are certain phenomena which tend to show that in many people the tissues lack normal resistance. It is a well-established principle that no part of a living creature can be maintained

in a healthy condition when it ceases to perform its appointed functions, and, beyond doubt, under modern conditions the normal functions of the teeth and surrounding tissues are not performed. The consequence is that the tissues are deprived of a normal blood supply and their vitality is diminished. As the bone surrounding the teeth is a tissue of transient structure, depending for its very existence on the teeth themselves, it is only reasonable to assume that the growth of the bone tissues is very largely dependent on the functional activity of the teeth. Modern diet is such that the full functional activity of the teeth is seldom brought into play, much of the food requiring very little mastication, and this loss of the function of the teeth must seriously affect the structure of the bone forming the alveolar process and render it less resistant to attack. Clinical observation supports this view. In mouths which show that the function of mastication is duly performed the alveolar process is well developed, and if periodontal disease attacks such mouths its progress is not rapid. On the other hand, if mastication has been imperfectly performed, the bony coverings of the teeth are thin and sparse, and the disease, when once started, progresses rapidly.

There is another point to be considered in connection with the resistance of the tissues of the bone; viz., that any inimical condition affecting the osseous system is likely to show itself in the alveolar process at an early age, the reason being that, as already explained, the alveolar process is a transient structure.

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CHAPTER V.

The Etiology of Periodontal Disease.

CONSIDERABLE diversity of opinion exists as to the cause of chronic general periodontitis.

In reviewing the pathology of the disease in animals it has been shown that the lesion was caused by injury from the food. When cats and dogs are fed on a diet of fresh flesh they are invariably free from the disease, but as soon as soft pap food is substituted the animals become a prey to the disease.

In the horse the disease can clearly be traced to injury from the food, the particular form in which the food is given being responsible for the injury. Under natural conditions horses feed upon grass which, being torn up in long strands and in a pliable state, can be masticated and disposed of without injury to the soft tissues. Under "civilized" conditions horses frequently cannot be put out to grass and the food is often given in the form of hay and straw (chaff) cut up into short lengths. Chaff in short stiff lengths is very likely to cause injury to the muco-periosteum through being pressed into spaces between the teeth. This view of the causation of the disease in horses is supported by the fact that the disease is prevalent wherever the conditions are such that fragments of thorn become mixed with the food. Similarly, as previously mentioned, cattle in parts of America are subject to the disease owing to the penetration of the tissues by the barbed crowns of *Hordeum jubatum*.

In the horse the disease is clearly to be attributed in a very large measure to alteration in the physical character of the food.

In man, too, the evidence points to the disease being started by injury of the gingival margin from food débris, or the local action of toxins as seen in the marginal gingivitis of mouth-breathers.

The prevalence of the disease is probably due to the character of the diet of the present day. Much of our food is now prepared in such a manner that it readily accumulates around the teeth and is of a character which easily undergoes fermentation. The food thus prepared requires but little mastication, with the result that the tissues in and around the teeth are deprived of an adequate blood supply and are thus rendered less resistant to attack. Evidence in favour of this is shown in a series of skulls of Londoners of the sixteenth and seventeenth centuries. Periodontal disease in the region of the molars is extremely common in the skulls, but the disease is chronic in character and the bone shows distinct reaction to injury. In the majority of these skulls the function of mastication was efficiently carried out as is shown by the attrition of the teeth. As soon as the disease has started, the formation of abnormal pockets favours the accumulation of débris, and so the action of the toxins is accelerated.

It is necessary here to consider wherein lies the difference between caries and periodontal disease, seeing that both are traceable to the lodgment of food. It is probable that the deciding factor is the predominance of particular foodstuffs. On carbohydrates enzyme action results in the production mainly of lactic acid, while on proteins enzyme action results in the formation of ptomaines which are alkaline in reaction.

If we turn to the food of modern times we notice a very distinct increase in the number of made-up dishes with a corresponding decrease of plain fresh-cooked food. A large proportion of the animal food is imported and has been kept in an ice-bound condition. Such food undergoes putrefactive changes more quickly than fresh animal food.

Attention has been drawn to the intimate relationship between periodontal disease and mouth-breathing, and the increase in the number of individuals who are mouth-breathers has, undoubtedly, added to the increase of the disease.

CHAPTER VI.

Periodontal Disease as the Active Agent in the Production of Pathological Lesions.

It is now generally admitted that periodontal disease may be the starting point of various other pathological lesions, and it is proposed, therefore, to review this subject broadly, and to make special reference to certain diseases which seem to be frequently traceable to mouth infection. A full and detailed discussion of this question would be beyond the scope of the present work and would necessitate too deep an incursion into the realm of general pathology.

(A) The Normal Condition of the Alimentary Canal.

In the normal mouth the teeth are arranged in a regular arch ; the gums are firm and of a light pink colour, and are attached to the teeth at their necks. In the process of mastication food tends to cling about the teeth, especially around the necks of the teeth where the gingival margin is attached in such a way as to leave a slight trough, but when normal conditions prevail the mouth is quickly freed of food débris by the combined action of the cheeks, tongue and saliva.

In the healthy mouth certain organisms are always present. The majority of these organisms cannot be grown in ordinary culture media, and, of those which can be grown, the streptococci are in "overwhelming preponderance." When food is being taken, and probably at other times, adventitious organisms are introduced into the oral cavity.

Various factors contribute towards the maintenance of the "bacteriological balance" in the mouth, e.g. (1) the mouth

is constantly flushed with saliva which carries the organisms to the stomach. (2) Phagocytes are incessantly passing up between the epithelial cells, and either take up the organisms or discharge their bactericidal content; some, "having performed their functions, undergo dissolution or are swept away by the currents of saliva," while others find their way back into the subjacent tissues and are rapidly destroyed. (3) The bacterial struggle for existence is probably a contributory factor as suggested by the results of Miller's experiments. In one experiment he thoroughly rinsed his mouth with a bouillon culture of *Bacillus prodigiosus* containing over 2,000,000,000 bacilli, and found that at the end of three hours his mouth was practically free of that organism.

The organisms reaching the stomach from the mouth are to a great extent killed or inhibited by the action of the gastric juice, and a few hours after meals the duodenum may be found quite sterile. In the small intestine bacterial growth starts afresh, the rate of growth increasing as the colon is approached; at the colon bacterial multiplication reaches its maximum. The bile exercises a selective action on the organisms, and, while checking the growth of certain organisms such as the delicate streptococci, the pneumococci and the *Bacillus proteus*, has no effect on the typical intestinal bacteria such as the *B. coli* group and the hardier streptococci. The bacteria in the intestines are disposed of in two ways. Some are expelled with the fæces, and these are probably the majority, while others find a passage through the intestinal wall. The latter are arrested either in the subcutaneous lymph nodules, or, evading these, are either trapped in the mesenteric and retroperitoneal lymph glands, or reach the venous radicles of the portal vein where the endothelial cells arrest the leucocytes with the contained bacteria.

With regard to the toxic products of the bacteria, it is, says Andrewes,[1] "conjectured that such injurious substances absorbed from the alimentary canal as may escape the alchemy of the

[1] Discussion on Alimentary Toxæmia, *Proc. Roy. Soc. Med.*, vol. vi, No. 5 (Supplement).

liver are neutralized by the secretion of certain of the ductless glands—for example, the thyroid."

Briefly, it may be said that under normal conditions the organisms in the alimentary canal are efficiently dealt with by the body defences and their presence causes no ill-effects.

(B) Abnormal Conditions of the Alimentary Canal.

Any septic condition of the mouth adds fresh organisms, the normal flora being increased and new varieties appearing. "The abnormal products of tissue reaction to injury are present, that is, inflammatory exudation which is rich in proteid, and an enormous increase of dead cells, epithelial and otherwise." The abnormal flora in conjunction with abnormal environment modify the virulence of the organisms in the direction either of attenuation or of exaltation.

The local effect of this increase of organisms is to undermine and weaken the epithelial covering, which then affords a passage for the organisms into the deeper tissues, and, in periodontal disease, where the organisms and their product are stagnant in the "pockets," the organisms constantly pass into the medulla of the bone and are absorbed into the general circulation. This latter condition is manifestly present where there is marked rarefying osteitis, and is probably also present in many cases where there is but little bone destruction. This question was referred to on p. 54.

The presence of an increased number of organisms and their toxins in the stomach generally results in a gastritis, which in its turn lessens the efficiency of the gastric defence, and an overwhelming number of organisms pass into the small intestines. Under these conditions the defences of the intestines are taxed to their uttermost, and a greater number of organisms pass from the intestinal surface into the system. In the opinion of Adami this condition often leads to a sub-infection rather than an active infection. "The bacteria carried in do not multiply and set up foci of suppuration; they are destroyed, but with their destruction the liberation of their toxins causes a poisoning

of the cells immediately around them, and the accumulative action of these toxins, whether locally or at a distance, upon the liver cells, for example, brings about the death of certain cells and replacement by fibrous tissue."

The toxic products of the bacteria are also increased, and, as the normal defences are unable to neutralize them, they pass these defences and are distributed to the tissues, attacking those cells for which they have the requisite chemical affinity. In this manner the organisms or their toxins are absorbed into the blood-stream either directly via the bone or indirectly via the intestinal canal, and the pernicious influence of periodontal disease thus passes deep into the system and starts other diseases.

The disease which results from the infection of the blood-stream varies in different individuals. The reason that the resulting disease takes different forms in different individuals is not far to seek and has been well explained by Dr. Stanley Colyer as follows : " In all diseases there are always two factors at least, the seed and the soil, without which disease cannot exist. The seed varies, the soil varies; never are the two the same ; never is the relation repeated. This is why variation in disease occurs, and in no two persons does it run a precisely similar course. Oral sepsis is but a comprehensive term to include various forms of septic conditions of the mouth, it is not a disease. The germs causing the sepsis vary, and so the germs passing into the body and the toxins absorbed produce different results in different people. It cannot be said precisely why germs of a particular kind entering one body produce a septic-æmia, and in another an infective endocarditis ; or why a toxin in one will produce anæmia, and in another a neuritis ; that such is the case, however, seems comparatively certain, and one must rest content for the moment with the fact."

Caution is necessary in assuming that periodontal disease is the cause of some other disease with which it is associated until all other possible sources of infection have been eliminated. But even where sepsis in the mouth is not the primary cause, it must not be neglected; otherwise, by lowering resistance, it will assist the pathological process.

(C) Diseases associated with Periodontal Disease.

Certain conditions seem to be closely connected with periodontal disease. Chronic inflammatory changes in the oropharynx, if not directly due to the persistent infection from the tooth sockets, are undoubtedly markedly affected by it. Amongst those conditions chronic glossitis is of special interest because of the marked tendency for this condition to pass into carcinoma.

Syphilis is usually regarded as the principal cause of chronic glossitis, but, where there is no dental sepsis, there does not appear to be any special tendency for syphilitics to develop chronic glossitis, whereas when dental sepsis is added chronic glossitis usually develops.

For years past it has been recognized in surgical practice that there is a very great tendency for carcinoma of the tongue to develop in patients with chronic glossitis and a history of syphilis. During the last three years cases in which the tongue began to show suspicious appearances and in which all sources of dental sepsis were then removed by extraction of the teeth have been watched, and in every instance the removal of the sepsis was followed by marked improvement. These facts certainly point to an intimate connection between dental sepsis and carcinoma of the tongue.

There is ample evidence to show that oral sepsis and gastric affections are closely connected. In many instances the gastric condition is the direct result of the constant presence of septic saliva, and cases are constantly occurring in practice in which, with the removal of the oral sepsis, the gastric condition completely clears up. In such cases the chain of events is probably as follows : The food, imperfectly masticated and incorporated with septic saliva, undergoes excessive fermentation, with the result that, sooner or later, a catarrhal inflammation of the gastric mucosa is started. The chronic gastritis is probably of the mucous variety in which the acidity is always slight, a point of considerable importance as far as the inhibition of the microbes is concerned. The catarrhal inflammation is kept alive by the constant presence of the septic matter, and may become infective in character. There is reason

to believe that sepsis from the mouth is the cause of ulcer of the stomach found in old people; but the gastric ulcer seen in young girls no doubt arises from some other cause, as, in a large proportion of the latter cases, the mouths are clean. But in all cases of gastric ulcer, whatever the cause, it is probable that healing would be retarded or prevented while oral sepsis is present.

As pointed out above, the gastric juice under normal conditions inhibits or destroys the majority of organisms entering the stomach, and consequently the number passing into the duodenum is relatively small. But the bactericidal power of the gastric juice is diminished in cases of gastritis and there is then an increase in the number of organisms passing through the pyloric opening, and a greater risk of infection in the small intestine.

The gastritis may be of an infective character, and a case of this kind is quoted by Dr. Hunter[1] who considered that the gastritis was distinctly traceable to the mouth condition. The history of the case was as follows:—

"This was a case of subacute gastritis in a woman, aged 62. The patient suffered from severe intermittent sickness and gastric pain, necessitating the use of morphia, lasting eight months, with loss of weight and increasing weakness. Cancer was suspected, but on examination no sign of malignant disease was visible in the stomach, the abdomen, the rectum, or the uterus. Constant complaint was made of a bitter taste in the mouth, nausea, and loathing and distaste for food. The vomit in this case was loaded with streptococci, staphylococci, and a few bacilli. The only teeth present were three roots, around which there was a free discharge of pus. With the removal of these teeth the gastric condition rapidly improved." In this case it would seem that the mouth condition was responsible for the infection of the gastric mucosa which, owing to persistent damage, had been deprived of its natural powers of resistance and so rendered susceptible to attack.

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[1] *Lancet*, January 27, 1900.

There is every reason to believe that a duodenitis may result from the increased number of organisms passing through the pylorus, and that the infection may spread along the common bile-duct and cystic duct to the gall-bladder. In this manner a chronic cystitis may be started, a condition which, pathologists maintain, is necessary for the formation of gall-stones. It seems probable that infection may spread along the pancreatic duct and give rise to a pancreatitis which will cause pressure on the common duct and thus lead to jaundice. This view is supported by the fact that, in many cases of jaundice, Cammidge's pancreatic reaction may be obtained. The passage of the toxic products from the stomach, in addition to starting a duodenitis, may lead to an enteritis and possibly colitis. P. Daniel [1] maintains that, in cases of oral sepsis, the lower part of the ileum is always in an infected state.

When a general gastro-enteritis has been established, toxic matter will be produced in abundance, which, on being absorbed, will initiate various general lesions.

To sum up briefly, it would seem that oral sepsis leads to gastritis, and that gastritis leads directly or indirectly to a general inflammation and infection of the intestinal canal. It is in the intestine that the toxins are produced in greater quantity, and it is from this area that they are mainly absorbed.

It is quite true that many patients under 40 years of age, who have suffered from periodontal disease for several years, may show no signs of gastro-intestinal disease; but, after that age, periodontal disease undoubtedly gives rise to gastro-intestinal trouble in a very large number of cases. The explanation is that up to the age of 40 the body resistance successfully combats the abnormal gastric condition, but after that age the body resistance gradually diminishes and infection results.

As the abnormal conditions started in the oro-gastro-intestinal canal by periodontal disease may be regarded as the result of direct infection, we will next consider some of the pathological conditions created by the continuous passage of the

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[1] *Lancet*, January 15, 1910.

bacteria into the tissues, or the absorption of toxins from the diseased oro-gastro-intestinal canal. One of the commonest of these conditions is that of "chronic ill-health." There is a lack of energy, a general condition of malnutrition; a sallow, unhealthy appearance in young people, acne pustulosa, and in elderly people acne rosacea and eczema, and, invariably, a history of gastro-intestinal disturbance. This condition should be regarded as the result of the continued absorption of small doses of toxins and should be classed as a chronic toxæmia. This toxæmia is probably due to: (1) Absorption of toxic products of organisms; (2) absorption of toxins produced from abnormal digestive processes. When the source of the oral sepsis has been removed there is a rapid improvement in the gastric condition and the general health; the improvement, be it noted, commencing before the insertion of dentures.

In these cases the extraction of the teeth not only eradicates the main source of toxins from the mouth, but also, by removing the cause of the gastro-intestinal condition, stops the formation and absorption of toxins from that area.

In practice there is a type of case met with in young adults in which there is a loss of weight, accompanied by a constant rise of temperature towards the end of the day, and the development of physical signs suggesting tubercle. An examination of the sputum fails to demonstrate the tubercle bacillus. I have seen several cases in which the symptoms have cleared up completely on the removal of the dental sepsis. The following is a typical case: A young man, aged 34, developed in October, 1913, an irido-cylitis, which was diagnosed as tuberculous. For some time previous to this he had been generally run-down, and, shortly after the eye lesion appeared he showed definite physical signs of early tubercle of the lung, with a rise of temperature every evening. Examination of the sputum failed to demonstrate the presence of the tubercle bacillus. He was sent away as a suspicious case for a prolonged rest by the sea. The medical man under whose care he was placed suspected that the eye lesion was septic, and he was referred to me to report on the

condition of the teeth. There was a fair degree of periodontal disease with marked gingivitis. The patient was a mouth-breather. Removal of the teeth was advised. With the extraction of the first batch, four in number, the lung symptoms, which had become quiescent, returned together with the evening rise of temperature and physical signs in the chest. When these had again subsided the teeth were gradually removed, and he made a rapid and complete recovery. The eye condition cleared up, and the general health showed marked improvement. In the following July he was said to be in better health than he had been for years, and the improvement was remarked on by his friends.

This case is of interest from the following points :—

(1) There was a marked family history of tubercle.

(2) The lung symptoms were extremely suspicious, and even if there was no active tubercle the presence of the oral sepsis rendered the patient much more liable to direct tuberculous infection. The necessity for a clean mouth was clearly indicated.

(3) The return of the lung symptoms after the removal of the teeth indicated a causal relationship.

This case, viewed by the experience of others I have seen, suggests that in individuals predisposed to tubercle the oral sepsis may act by lowering the general vitality, and so increasing the liability to tuberculous infection.

Those ill-defined conditions embraced by the terms "chronic rheumatism" and "muscular rheumatism" owe their origin to sepsis, and the focus of infection is frequently the mouth. The rapidity with which these conditions, often of long standing, will entirely clear up with the removal of the periodontal disease is truly remarkable.

The form of arthritis usually classified as rheumatoid arthritis is now generally accepted as having an infective origin. In this type of arthritis the larger and sometimes the smaller joints of the hand and foot are affected, the morbid process having its origin in the peri-articular tissues. It is usually met with in patients under 40 years of age, and is more common in women than in men, but it is seen about equally in boys and girls.

There is abundant clinical evidence to show that the focus of infection is frequently traceable to the mouth, and the rapid and permanent recovery which often follows the removal of the periodontal disease in these cases clearly shows the intimate connection between the two conditions. A word of warning, however, must be uttered against prematurely assuming that where periodontal disease and rheumatoid arthritis are associated they necessarily stand in the relation of cause and effect, as other active foci of infection may be present; for example, the vagina, urinary system, &c., which may be the main source of infection.

The Hunterian lecturer for 1899, Sir Douglas Powell, recorded a case of cardiac irregularity which was only slightly relieved by a course of Nauheim baths and resistance exercises, but which entirely ceased when the teeth had been treated. Cases of this character are by no means uncommon, the relief to the heart symptoms almost immediately following the removal of the septic foci in the mouth. The irregularity of the heart may be explained on the assumption of a toxin from the mouth, or gastro-intestinal tract, having a specific affinity for certain of the cardiac nerves.

Lastly, mention must be made of the importance of periodontitis as a cause of disease in the eye. This has been fully appreciated since it was recognized that a focus of chronic sepsis, situated in any part of the body, could give rise to an acute or chronic inflammation in any of the tissues of the eye, with the possible exception of the lens, which may suffer, however, in a secondary manner.

Amongst 215 cases of inflammation of the eye, which Mr. W. Lang[1] attributed to sepsis, 129 were caused by periodontitis. He found also that the more vascular parts, the iris, the ciliary body and the choroid, were affected seven times more frequently than the less vascular sclerotic, retina and optic nerve, and only in one instance was the cornea primarily attacked. It is probable, therefore, that the poison is carried in the blood-stream.

I am indebted to Mr. Lang for the following account of the relations of dental sepsis to eye lesions.

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[1] Supplement to *Proc. Roy. Soc. Med.*, May, 1913.

As the pathology of these eye affections is still obscure it is only possible to prove that these diseases of the teeth and of the eye bear the relationship of cause and effect by selecting cases where only one source of sepsis appears to be present, removing it and watching the result. In this way and without any other treatment recently lost sight has been rapidly regained, and acute attacks of inflammation have been cut short and have not recurred, whereas in former times, when the source of sepsis was not removed, the sight in similar cases was not restored in spite of every known treatment and the attacks of inflammation recurred until the eye was lost.

The following is a list of affections which periodontitis may cause :—

Interstitial keratitis.

Scleratitis and episcleritis.

Iritis and irido-cyclitis with exudation into the vitreous followed by secondary cataract and secondary glaucoma.

Central choroiditis, which was formerly described as senile central choroiditis and was attributed to senility when the patient was elderly, though similar disease occurs in young people, but not so frequently.

Disseminated choroiditis, confluent and discrete, which may be followed by detachment of the retina.

Choroido-retinitis.

Optic neuritis. Progressive myopia and asthenopia.

Until quite recently these diseases were attributed either to gout or to rheumatism, whenever tubercle, syphilis, gonorrhœa, diabetes, albuminuria and fevers could be excluded. It is now known that they are more frequently due to some septic infection in the proportion of thirteen of septic to ten of all other kinds. The following instances illustrate this view :—

Episcleritis.—A patient with this condition which had been present for five months recovered after treating the periodontitis. It recurred again and after lasting a year all the teeth were removed. The next day the eye was whiter. The general health improved and she said "life was worth living," and the eye recovered rapidly. Two years later she had appendicitis and the

episcleritis returned; both diseases quieted down together and six years later neither had relapsed.

Kerato-iritis with Vitreous Opacities and Secondary Glaucoma.—A man, aged 37, with normal vision $\frac{5}{5}$ in each eye, had mutton-fat keratitis punctata and periodontitis, for which he was treated. A year later, when he was seen by another surgeon, he could only count fingers at two feet with the right and at one foot with the left eye. The vitreous was too hazy for the fundus to be seen, the tension was raised and the field of vision contracted in the right eye. Another dentist condemned all the teeth and when they had been removed the vision began to improve. Three years later the vision in each eye was $\frac{5}{5}$ and SN 0·25'.

Central Choroiditis. — An officer of the Yeomanry, who thought himself to be in good health, noticed two days before coming under observation a brown stain on the newspaper. On covering one eye he observed a blur which involved the fixation point; this was found to be due to a small patch of central choroiditis which had reduced the vision to $\frac{5}{6}$ slowly. Five days later the brown mist had gone and the vision was $\frac{5}{5}$. In the interval three septic roots had been removed and his periodontitis treated.

Kerato-iritis, Secondary Cataract and Secondary Glaucoma.— A lady, who had suffered from recurring attacks of inflammation in both eyes for fifteen years, during which one eye had been excised and a secondary cataract removed from the other, came under observation for attacks of glaucoma, which obscured the sight every morning, but cleared again at night, when she saw very well. In this state of obscuration which reduced the sight to the counting of fingers at a distance of three feet, all her septic teeth were removed under a general anæsthetic. On regaining consciousness the vision was clear. A year later it was reported to be normal and never to have been again obscured.

Disseminated Choroiditis. — In this patient disseminated choroiditis in one eye had reduced the vision in six months to $\frac{6}{60}$. She had all her teeth removed for periodontitis. In two days the vision was $\frac{6}{9}$; three weeks later it had gone

back to $\frac{6}{24}$; a root which had been overlooked was removed the same day and within a few days the vision returned to $\frac{6}{9}$.

Operation for Senile Cataract.—A lady with a septic mouth, which she refused to have treated before an operation for senile cataract, submitted to having all her teeth removed, when an iritis began to cause pain four days after the cataract had been extracted. The same day the pain in the head and eyes was less, and the inflammation ceased to increase; two days later the gums, which could not be cleaned up before the teeth were removed, were foul, and the eye was worse. In a few days the gums were clean and at the same time the eye improved and recovered very quickly and was well within three weeks. In similar cases where the source of sepsis is not removed the eye would remain inflamed for many weeks.

It is not intended that the foregoing should be regarded as dealing exhaustively with the special conditions which are traceable to periodontal disease. Medical literature of recent years contains numerous references to other conditions having a similiar source. But sufficient has been said to demonstrate that sepsis around the teeth is a potent source of general infection, and that wherever a disease has a septic origin dental sepsis may be the cause, or at any rate a contributing cause. It is a little difficult to understand why medical men should so often place the utmost importance on a small area of sepsis in other parts of the body and refuse to recognize the serious source of sepsis so often present in the mouth.

CHAPTER VII.

The Treatment of Periodontal Disease.

In the chapter on the morbid anatomy and pathology of the disease, it was pointed out that the formation around the teeth of "pockets," which inevitably become stagnation areas, is the outstanding feature of the disease. Until these "pockets" or stagnation areas have been eradicated, it does not seem reasonable to claim that a cure has been effected, and as it is almost impracticable to eradicate them actual cures of periodontal disease are rare, though well-directed treatment may keep the disease in check. Reference has also been made to the important rôle which mouth-breathing plays in the promotion of the disease. It has also been shown that the progress and severity of the disease is greatly influenced by the degree of resistance offered by the tissues.

Treatment, therefore, should be directed towards promoting efficient drainage, raising the resistance of the tissue and ensuring proper nasal breathing.

With regard to the breathing, I am firmly convinced that in all cases of mouth-breathing, even if the patient is addicted to this habit for a few hours a day only, there is very little hope of checking the disease; still further, in such cases the general symptoms seem more pronounced. It is essential, therefore, to ensure proper nasal breathing. Where the patient breathes by the mouth the nasal cavity must be examined and any obstruction removed. Some patients persist in the habit of mouth-breathing after a free nasal passage has been obtained. The habit can, however, often be overcome by the use of the apparatus suggested by Mr. W. W. James and shown in fig. 80.

The therapeutic measures to be adopted may be divided into (I) Local and (II) General.

(I) Local Treatment.

Local treatment consists in the promoting of asepsis as far as practicable by securing efficient drainage. Efficient drainage relieves the tissues of the constant injury arising from the toxic matter around the teeth and the tissues react more readily. It may be confidently affirmed that the more thoroughly the pockets are kept free from sepsis, the greater will become the prospects of staying the progress of the disease. In theory efficient drainage would seem an easy matter to establish; in practice, however, such is not the case. The local remedies employed should be directed towards—

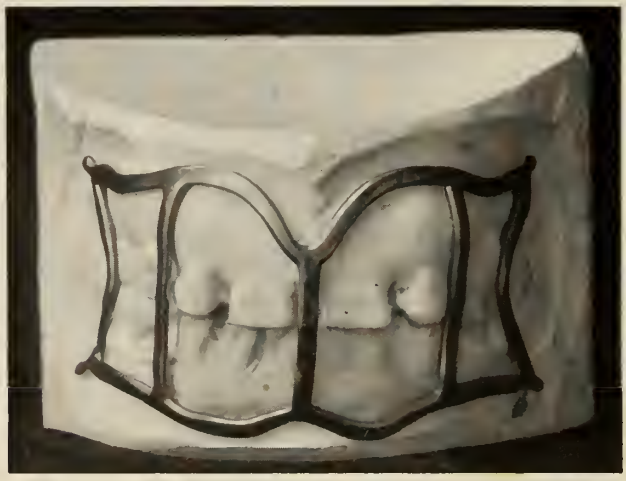


Fig. 80.

(*a*) Freeing the teeth from salivary calculus and other débris.

(*b*) Reducing the depth of the pocket.

(*c*) Cleansing the pocket.

(*d*) Massage of the gums.

(*a*) The removal of salivary calculus allows the pockets to drain more freely and facilitates irrigation of the pockets. The

scaling must be done thoroughly and special care taken to remove any calculus in the interstitial spaces. Where there is much gingivitis it is advisable to delay the scaling until the gingivitis has been reduced by appropriate treatment, and the general resistance raised by improving the general health. The operation of scaling can then be carried out more efficiently and with less laceration of the tissues.

(*b*) In considering methods for reducing the depth of the pockets, it must be remembered that the pockets are deepest at the most inaccessible parts, *i.e.*, in the interspaces. The regular use of astringents will often produce good results. Tannic acid used in the form of a powder may be adopted and should be rubbed into the gums for two minutes once a day, the treatment being continued for at least two months. If at the end of this period the gum margins have shrunk the application may be reduced from once a day to twice a week. As tannic acid is liable to leave an indelible stain on linen, patients using it should be warned against wiping their hands on clean linen.

The free removal of the redundant gum with scissors and small knives is an excellent method of reducing the depth of the pocket, especially where there is considerable destruction of the membrane on the labial aspects. The actual cautery is another useful method of destroying the gum margins ; this method is especially useful in the interproximal spaces.

(*c*) Regular cleansing of the pockets and interproximal spaces by the patient is the essential part of the local treatment. Firstly, the spaces between the teeth must be freed of food débris by means of "floss silk," and the gums should then be well squeezed between the thumb and the first finger, with a downward pressure on the upper teeth and an upward pressure on the lower teeth. The next step is to "irrigate the pockets." If the irrigation can be easily carried out no special fluid is necessary and a free flushing with sterilized water would be effective. The most efficient method is to flush the pocket with peroxide of hydrogen (vols. xv), using a hypodermic syringe. The needle of the syringe is passed well into the pocket and gentle pressure exerted. The use of force is to be deprecated

as it is essential that any granulation tissue that may be present should not be broken down. But as a hypodermic syringe cannot be readily manipulated by the very large majority of patients it is generally necessary to try an easier, if less efficient, means of irrigation. I find the best plan is to have the pockets wiped out with a wisp of cotton-wool dipped in hydrogen peroxide, the cotton-wool being wound round a fine broach. It is necessary to give patients very precise instructions. The broach should be of the type ordinarily used for root canal dressings. The method of winding the wool on the broach should be demonstrated. I would suggest the following plan : Spread a wisp of cotton-wool along the palmar surface of the terminal phalanx of the first finger of the left hand ; place the broach on the centre of the wool ; fold the cotton-wool over the broach ; bring the thumb on to the broach and then run the thumb with the broach up the palmar aspect of the first finger ; the fragment of wool over the point should then be turned down so as to prevent the point sticking through.

Such detailed instructions may seem hardly necessary, but experience has taught me that the success of this method of treatment depends in a large measure on the skill of the patient in twisting the wool on to the broach. The patient should be shown exactly where and how to apply the peroxide, and instructed to pay special attention to the irrigation of the spaces between the posterior teeth. The hydrogen peroxide to be used should be poured into a small receptacle, and any left unused should be thrown away and not returned to the bottle. The pockets should be cleansed at least once a day, the best time being shortly before retiring for the night.

If the patient will only carry out this method of cleansing the spaces thoroughly and regularly, a marked improvement may be confidently anticipated.

(*d*) *Massage of the gums* by regular rubbing with the fingers and with the tooth-brush will assist in improving the condition of the gum margins.

(II) General Treatment.

General treatment consists in raising the resistance of the tissues and may be considered under the following headings :—

(1) *Hetero-inoculation* (vaccine therapy).

(2) *Auto-inoculation.*

(3) Treatment directed towards the removal of systematic intoxication and a general improvement in health.

(1) *Hetero-inoculation, Vaccine Therapy.*—This method of treatment aims at assisting the tissues to defend themselves against the action of bacteria and their products. The method of procedure suggested by Eyre[1] is as follows : "The patient is instructed to grasp the lip opposite the affected teeth with the forefinger and thumb of each hand and draw it away from the gums ; in some cases a small roll of absorbent cotton-wool is packed into the sulcus between the alveolar process and the lip. The gum margin is next wiped with a sterile swab of cotton-wool mounted on the end of a stick, and the gum itself dried with a second sterile swab. Then with another swab firm pressure is made on the gum over the root of the tooth. The first drop or two of pus that exudes is mopped up with a third sterile swab and the pressure continued, and the pus that next exudes is collected on still another sterile swab, or by means of a stout platinum needle. This pus is employed for the purpose of making coverslip films. Finally, more pus is expressed in a similar way and used to inoculate tubes of nutrient media."

The most satisfactory method is to extract a tooth when this is practicable, for preference a molar, and take a culture from the space between the roots or from near the apex of the tooth. Pure cultures of the various micrococci present are then obtained *secundum artem.* The patient's serum is next examined with a view to obtaining evidence of the presence of antibodies to some or any of the organisms isolated which would indicate actual and active infection. Opsonins, amboceptors, and agglutinins are the most useful antibodies in this connection, in the order mentioned. The organism towards which a low index (0·5 or

[1] *Proc. Roy. Soc. Med.* (Odont. Sect.), vol. iii, p. 34.

lower) or a high index (1·3 or higher) is recorded is usually regarded as the possible origin of infection and from this organism a vaccine is prepared. (Mr. Goadby suggests that if more than one organism shows a low index a mixed vaccine of the several organisms should be obtained.) If two or more organisms are associated, as is frequently the case, a separate vaccine should be prepared of each, and if subsequent observations of the index towards each bacterium are recorded, the dosage of either vaccine may be modified as necessary. In all cases, and particularly if improvement is slow, further bacteriological examination of the case should be made from time to time.

The vaccines are prepared in doses of varying strength, each dose being placed in a small sterile glass bulb. The dose depends upon the responsible organism. Dr. Eyre recommends doses as follows for the most commonly encountered infecting micro-organisms :—

	Millions									
Micrococcus catarrhalis if apparently the sole infecting organism	5	...	10	...	25	...	50			
Micrococcus catarrhalis associated with streptococcus	10	...	25	...	50	...	75	...	100.	
Micrococcus paratetragenus	10	..	25	...	50					
Micrococcus pyogenes aureus	50	...	100	...	250	...	500			
Streptococcus lanceolatus pneumoniæ (with which *Streptobacillus malæ* (Goadby) is probably identical)	5	...	10	...	25	...	50			
Streptococcus pyogenes longus	5	...	10	...	25	...	50			
Bacillus pneumoniæ (Friedländer) ...	5	...	10	...	50	...	100			
B. pyocyaneus	10	...	50	...	100	...	250			

The injection of the vaccine must be carried out under strictly aseptic precautions.

(2) *Auto-inoculation.*—D. E. C. Hort[1] has drawn attention to the fact that in vaccine therapy the damage to the tissues by the toxic products of autolysis and other cell metabolism is not taken into consideration. These toxic products are combated by the formation of corresponding antibodies. Any method of treatment which produces hyperæmia in the part, whether by the use of poultices or blisters or Bier's treatment, acts by

[1] *Proc. Roy. Soc. Med.* (Med. Sect.), vol. ii, No. 6.

increasing the fluid exudate in the affected parts; in this way the irritant is diluted and the natural antibodies increased in the area; in other words, the treatment may be regarded as a process of auto-inoculation.

The ideal method of producing hyperæmia would seem to be that suggested by Dr. Bier, but it presents some practical difficulties. To carry out the treatment a splint is made to grasp the gums as high above the teeth as possible (fig. 81). The spring should be so adjusted as to cause sufficient pressure to congest the gums without causing pain. The splint should be applied for about fifteen minutes twice a day.

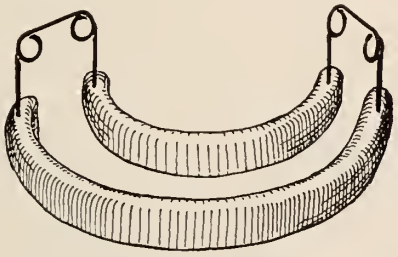


FIG. 81.—Semi-diagramatic view of splint.

A useful apparatus for producing hyperæmia of the gums has been suggested by Mr. H. Woodruff. A vulcanite cap is made to grasp the margin of the gums as high above the tooth as possible, the remaining portion of the cap being made so that a slight space exists between the gums and the splint. A tube is inserted into the splint and connected with an exhaust bulb. The apparatus is shown in fig. 82. The cap is placed over the teeth and the air is gradually exhausted. By this means an effective hyperæmia is obtained. From a practical point of view the results from this line of treatment have been disappointing.

The nearer the health approaches to the normal standard the greater becomes the resistance of the tissues and their power to react. An impairment of the general health means a lowering of the resistance of the tissues and of their power to react; it is, therefore, very important that steps should be taken to improve the general health as far as possible. This part of the treatment,

however, belongs to the province of the general medical attendant.

The question of treatment must now be considered more in detail, and it will be convenient to discuss separately the cases which are favourable for treatment and those which are not.

Fig. 82.

(1) Cases favourable for Treatment.

A case may be regarded as favourable for treatment if the following conditions are found :—

(*a*) The pockets around the teeth are shallow.

(*b*) The arch is well developed, and the function of mastication is efficiently performed.

(*c*) There are indications of recuperative powers on the part of the patient.

(*d*) The patient is a nose-breather.

As the pockets, which are the real cause of the trouble, cannot be removed, treatment resolves itself into a question of drainage. The more thoroughly the pockets are cleansed, the

greater will be the chance of staying the progress of the disease. The teeth must be thoroughly scaled, and the patient instructed to carry out the following daily routine :—

(*a*) Pass silk between the teeth to free the spaces of food débris.

(*b*) Squeeze the gums so as to expel as much material as possible from the pockets.

(*c*) Brush the teeth and gums thoroughly with a stiff brush.

(*d*) Irrigate each space with peroxide of hydrogen, as suggested on p. 79.

(*e*) Twice a week apply to the pockets a 2 per cent. solution of iodine in alcohol.

These simple measures, if faithfully carried out, will suffice to arrest the disease in favourable cases. I have tried raising the resistance of the tissues by means of vaccines and Bier's method of congestion, but I am not satisfied that these cases showed greater improvement than others where the treatment was limited to irrigation.

Thorough cleanliness of the "pockets" is the keynote in treatment, and if this is achieved an arrest in the progress of the disease may be expected. The following cases may be quoted as examples :—

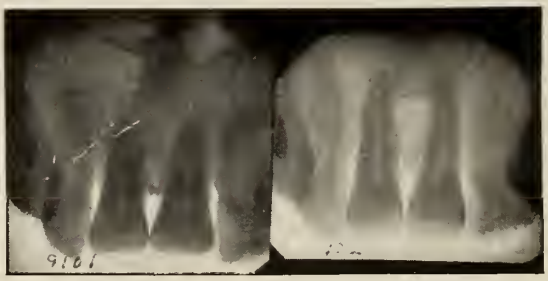
M. B. This patient, a well-developed female, was seen early in 1910. A well-marked gingivitis was present, with a slight thickening of the alveolar process, shallow pockets around the teeth, and a fair degree of attrition of the teeth.

The patient was a nose-breather. She was suffering from indigestion and rheumatism. Local treatment on the lines indicated above was adopted. The patient has lost all her general symptoms, and her mouth is healthy. Skiagrams taken before and after treatment show that in this case the condition of the bone is stationary (figs. 83 and 84).

This patient was last seen in October, 1915. She was still carrying out the treatment, and the condition of the alveolar process was practically similar to that shown in the diagram taken in October, 1911.

F. S. This patient was a female, well developed, and was first

seen in the latter part of 1909. The gums were as shown in fig. 85. There was marked gingivitis, but no apparent general symptoms. Skiagrams showed a fair amount of bone destruction. The patient was a partial mouth-breather, breathing by the mouth during sleep. Vaccines were tried without any



Taken October, 1911.

Taken February, 1910.

FIG. 83.—The light appearance of the film taken in February, 1910, is due to over-exposure.

Taken February, 1910.

Taken October, 1911.

FIG. 84.

irrigation of the tooth pockets. The condition showed no improvement, the discharge continuing from the gum margin. Local measures on the lines suggested were adopted, and the patient has very faithfully carried out her part of the contract.

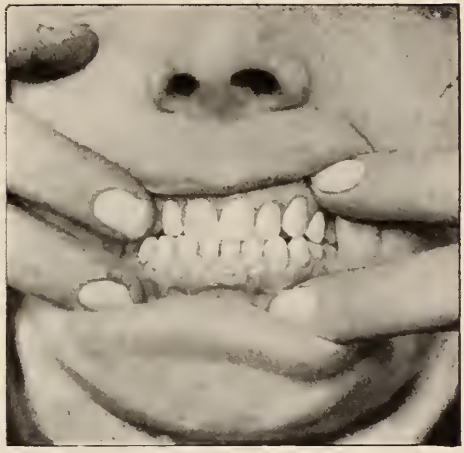
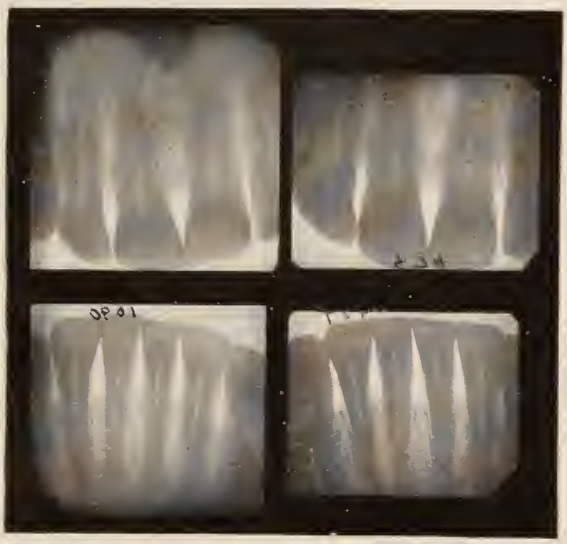


FIG. 85.

b



a

FIG. 86.—Skiagrams: (*a*) taken December, 1909; (*b*) November, 1911.

In this case the disease is progressing very slightly. Skiagrams taken at the commencement of treatment, and in November, 1911, are shown in fig. 86.

When last seen the disease was slowly progressing in spite of regular treatment carried out by the patient. The continued progress is to be attributed to the fact that the patient is a partial mouth-breather.

H. J. This patient came under treatment in December, 1908, suffering from suppuration in the left antrum and marked periodontal disease. The antrum was opened on December 23, and treated by lavage. Rapid improvement followed, but the discharge did not wholly clear up. A vaccine of *Staphylococcus albus* was used, and this treatment appeared to arrest the antral suppuration completely. A slight mucoid discharge occasionally appears, and the patient prefers to undertake the regular irrigation of the antrum rather than submit to a more extensive operation via the nasal fossa.

The periodontal condition was treated by scaling and irrigation of the tooth pockets. Bier's treatment was adopted for the anterior upper and lower teeth, the splints being used twice daily fifteen minutes at a time. At the end of 1909 the gum condition had considerably improved, and the patient's weight increased from 10 st. 2 lb. to 11 st. 12 lb.

Since then the treatment has been continued at intervals. Skiagrams show that the periodontal disease is practically stationary.

(2) Cases not favourable for Treatment.

Cases which are not favourable for treatment are those showing well-marked signs of rarefying osteitis and with general and local conditions, which indicate that the tissues have little recuperative power; also all cases of persistent mouth-breathing. It will be convenient to group these cases under two separate headings, viz.: (*a*) Cases unassociated with apparent symptoms; and (*b*) cases in which a causal relationship to other diseases has been established.

(a) Cases Unassociated with Apparent Symptoms.

As regards cases falling under this heading, there are certain points which must be kept clearly in mind in endeavouring to arrive at the right line of treatment. Although efficient irrigation may check the discharge from the tissues, and some advantage may be gained in an attempt to raise the resistance of the tissues by the aid of auto- or hetero-inoculation, all such efforts will fail to stop the advance of the disease. Under these circumstances the question arises whether it is justifiable to continue a course of treatment which, at the best, can do no more than slow down the progress of the disease. My own view is that such a course of treatment cannot be justified for two reasons: (1) Because a potential source of infection remains which may become active at any moment, and (2) because the destruction of the alveolar process is proceeding and thus increasing the difficulty of providing artificial dentures which will be efficient and comfortable.

The following case illustrates the first point :—

F. O., a female, aged 27, was first seen in July, 1909, and came under treatment on account of looseness of the teeth. Beyond slight indigestion she appeared to be in good health. The patient was a mouth-breather. A blood count taken showed a normal condition. The skiagrams indicated considerable rarefying osteitis (see fig. 87).

Extraction was advised, but was deferred at the earnest request of the patient, and an endeavour made to treat the condition locally. Three teeth were removed and local treatment and vaccines were tried. When seen in October, 1910, the condition of the mouth had improved and the teeth were firmer. The patient did not return again until February, 1911, and stated that she "had got tired of local treatment." Her condition was as follows: The skin was blotchy; her periods were irregular; she was suffering from gastritis, and complained of feeling thoroughly ill. A blood count showed well-marked anæmia and leucocytosis. The following teeth were removed :—

			6	5	4	3	2	1	1	2	3	4	5		
8					4	3	2	1	1	2	3	4	5		8

The patient recovered completely. This case is typical of many met with in practice. Local treatment is carried out at first, but is dropped gradually, and the mouth condition becomes an active focus of infection.

The second reason for early removal of the teeth in cases where the disease is making progress is also of practical importance. The progress of the disease implies destruction of the alveolar process, and eventually results in the complete disappearance of the alveolar process. From the prosthetic aspect it is then necessary to deal with a mouth devoid of ridges on which dentures can be steadied. Such cases are obviously unsatis-

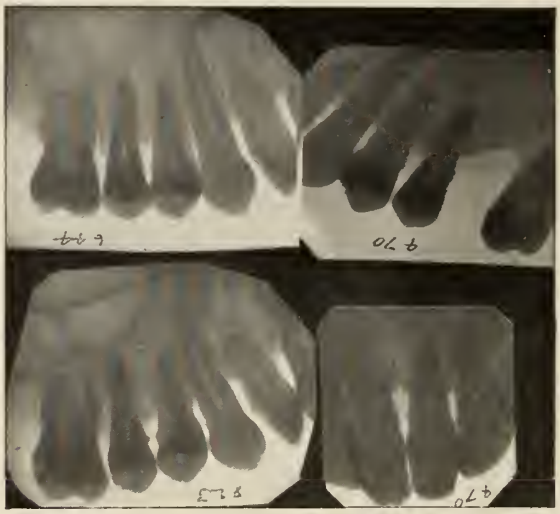


Fig. 87.

factory both to the practitioner and to the patient. If the teeth are removed at an earlier stage while there is ample alveolar process on which to rest the denture, the insertion of the denture will restore function to the bone, and, in most cases, a well-marked ridge will remain permanently.

For the foregoing reasons I have no hesitation in advising the removal of the teeth.

The following case is extremely interesting in its bearing on this problem. The models marked (*a*) in figs. 88 and 89 were

taken at the age of 40, and those marked (*b*) at the age of 75. The history of the case is briefly this: The premolars and molars had become loose and had fallen out, and as there was nothing to be gained by retaining the remaining teeth—sixteen in all—they were removed, dentures being inserted. If the models are carefully examined, it will be noticed that, where the teeth were

a *b*

FIG. 88.

a *b*

FIG. 89.

lost by the natural cure of the disease, the alveolar process has disappeared, but where the teeth were removed by extraction a well-marked ridge still exists. The early removal of the teeth resulted in the patient having for thirty-five years not only a clean mouth, but also dentures which have been steady and efficient.

(b) Cases in which a Causal Relationship to other Diseases has been established.

In this class of cases it is absolutely necessary that the dental sepsis should be completely removed. After reviewing the cases which have come under my notice during the last ten years, and careful consideration of the results of different methods of treatment, I am driven to the conclusion that the removal of the affected teeth at the earliest opportunity is the right course to adopt, and that any other line of treatment is unsatisfactory. A typical example of this class of case is as follows: A young adult is suffering from rheumatoid arthritis, and the mouth shows signs of general periodontal disease accompanied by rarefying osteitis in a marked degree. The case is one in which the potential source of trouble—that is, the pockets—cannot be removed by treatment. The choice here lies between retaining the teeth and running serious risk of an aggravated arthritic condition on the one hand, or, on the other hand, removing the teeth and ensuring useful joints. In my opinion, the latter course alone can be justified, as artificial dentures can be provided, but not new movable joints.

In carrying out extraction in these cases the following course is adopted: The mouth is made as healthy as possible by thorough irrigation of the pockets with hydrogen peroxide. The premolars and molars are next removed and the mouth is allowed to heal. Models of the mouth are then taken with the incisors in place, and bites are obtained. The advantage of proceeding on these lines is that you avoid the difficulty which would be experienced in gauging the correct height of the bite after the removal of the front teeth. The anterior teeth are then removed and dentures inserted as soon as possible. The number of teeth which can safely be removed at one sitting depends largely upon the individual case. Where the "power of repair" is at a low ebb the extractions must be carried out by easy stages, but where there is ample "power of repair" the extractions may be carried out more expeditiously. I incline to the view that extensive extractions at one sitting should be

avoided unless there are special reasons for adopting that course. It is said that trouble in the bone is likely to follow extensive extractions in these cases unless the resistance of the tissues is first raised by a course of vaccine treatment, but that is not my experience. I am disposed to think that, where trouble in the bone occurs as a result of extractions, it is almost always due to the damage inflicted on the tissues by the operation. A rise in temperature seldom follows the extractions if the mouth is kept clean. Occasionally a rise in temperature does occur, as is well shown in fig. 90. In that case the patient was suffering from a corneal ulcer, and the removal of each batch of teeth was followed by a rise in temperature.

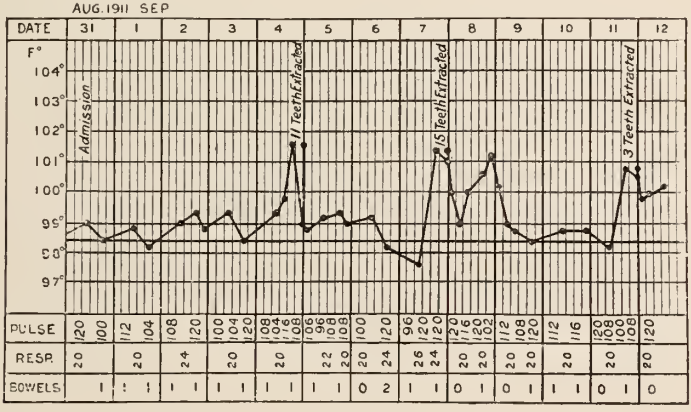


FIG. 90.

In cases where general trouble is traceable to the mouth it has been my practice to retain the anterior teeth, provided that the bone destruction is slight. The argument in favour of retaining the teeth in these cases is that the pockets around the anterior teeth are easily accessible and can be thoroughly irrigated by the patient. Experience of these cases has taught me, however, that if the patients are mouth-breathers it is only a matter of time before extraction becomes imperative. In this connection the following case is instructive. The premolars

and molars are removed for a patient suffering from naso-pharyngeal trouble. The remaining teeth were faithfully treated by the patient and the gums appeared to be in a fairly normal condition. At the end of four years, notwithstanding the care bestowed on the teeth by the patient, the bone destruction was progressing rapidly and there was a large amount of trouble around the teeth, necessitating their removal. Would it not have been a sounder policy, and much safer, if these teeth had been removed when the original extractions were carried out?

I am well aware that treatment by free extraction of all teeth which are the source of sepsis, as suggested above, is opposed to the teachings of many practitioners. With those who hold that local treatment will often result in a disappearance or at least an improvement in the general condition I fully agree, but —and this is the important fact—the source of sepsis has not been eradicated. The sepsis may possibly be lessened in degree, but nevertheless it remains and must continue to damage the tissues slowly but surely. The damage to the tissues is progressive, and when, through the loss of the teeth, a natural cure of the teeth is effected, the damage of the tissues may be irreparable. Dental practitioners cannot be justified in allowing such a condition to be brought about, and it is our duty to place clearly before the patient the risks they run.

Let us take as an example a patient who has been attacked for the first time with rheumatoid arthritis (polyarthritis), the toxic origin of which has been traced to the teeth. Here the following considerations should influence our decision as to advising free removal of the teeth :—

(1) Each fresh attack increases the injury to the joints and lowers the power of reaction of the tissues.

(2) Every tooth with a marginal gingivitis is a potential source of infection.

(3) The absorption of the toxins in these cases is often via the bone, skiagrams usually showing a fair amount of rarefying osteitis.

(4) Vaccine, ionic medication and local measures do not remove the real cause, namely, the pockets around the teeth,

and the patient, even if the condition has improved, is still left with potential sources of disease which may at any time set up a fresh attack of arthritis.

(5) The results following complete removal of the teeth are permanent, providing there are no other sources of infection.

Having discussed from a general point of view the treatment of periodontal disease, we may with advantage consider the important question of the value of vaccines.

Vaccine therapy aims at assisting the tissues to defend themselves against the action of bacteria and their products. It is essential for success, therefore, that we should know the causative organism of the disease which has to be treated. There is reason to believe, however, that periodontal disease is not caused by any special organism ; but even if it is due to a specific organism, that organism has not yet been identified. In the treatment of periodontal disease vaccine therapy therefore fails to satisfy the most important requirement. It may be urged in favour of vaccine treatment that the infection in the pockets is causing local injury, and that vaccines will check the injury by raising the resistance of the tissues in the neighbourhood of the tooth. The difficulty here lies in the fact that the infection is invariably mixed, and the vaccine therapist must needs prepare a vaccine of all the organisms found. The practice, however, is to use a vaccine of the predominant organism or perhaps of two of them, and, under these conditions, the treatment is necessarily incomplete. But, granting that vaccine treatment is occasionally successful, the pockets still remain, and no amount of vaccine treatment will remove them.

Apart, however, from any considerations which may be regarded as more or less theoretical, the question arises whether vaccines lead to good results in the treatment of periodontal disease. I have had personal experience of nearly forty cases of periodontal disease which were treated with vaccines, and in no single case was a cure effected and in only a few cases could I detect any improvement.

When vaccine treatment is carried out concurrently with local remedies and improvement results, it is extremely difficult

adequately to assess the amount of improvement which should be ascribed to the local measures on the one hand and to the vaccines on the other. It is the general experience that the majority of cases treated with energetic local measures will show a rapid improvement.

In papers advocating the treatment of periodontal disease by vaccines, one constantly reads that a large percentage of the cases are labelled as cured. Manifestly much depends upon the exact meaning placed by the practitioner on the word "cured," and reference to this point has already been made (see p. 77). Eyre and Payne[1] do not take the view there expressed, but class as "cured" all cases where "the affected teeth are firm so that mastication is painless and the mouth comfortable, no pus can be expressed from the sockets, the pains and other symptoms of ill-health have disappeared, and the patient feels 'quite well.'"

In such cases the patients are, in my opinion, relieved only and not cured, and to lead them to think that they are cured is to invite them into a "fool's paradise." The pockets remain and recurrence of trouble is only a matter of time.

In the treatment of the general condition arising from mouth infection vaccines may undoubtedly be extremely useful. The following case illustrates the occasional value of vaccines in the treatment of diseases which owe their origin to mouth infection:

A patient, aged 36, was seen on January 27, 1913. He was a carpenter's labourer, and since August 1912 had been under treatment for iritis in the right eye. There was no history of syphilis. The incisors and first molars were hypoplastic and there was very marked periodontal disease. He weighed ten stone. The following teeth were removed :—

		5	4	3	2	1	1	2	3	4	6	7	
8	7	5	4	3	2	1	1	2	3	4	5	6	7

By the middle of February all the teeth had been removed and a culture of streptococcus was obtained from the discharge between one of the maxillary molars. A vaccine was prepared,

[1] *Proc. Roy. Soc. Med.* (Odont. Sect.), vol. iii, p. 39.

but, owing to the fact that the patient was undergoing tuberculin treatment, it was not used until the end of April. The eye lesion, although slightly better, did not clear up with the tuberculin injection and it was not until streptococcal vaccine was used that any marked improvement was noticeable. The vaccines were stopped early in June when the eye condition showed considerable improvement. The patient was again seen in October. He had gained nearly one stone in weight, the inflammatory trouble had cleared up, but unfortunately the sight of the eye had been lost. The points to be noted in this case are :—

(1) There was no improvement in the eye condition under local remedies.

(2) The removal of the teeth, although improving the general condition of the patient, did not lead to any definite improvement in the eye condition.

(3) There was no apparent improvement with the tuberculin.

(4) Rapid improvement followed the use of the streptococcal vaccine.

The inferences to be drawn from these facts are that the eye lesion was due to infection in the mouth, and that, although removal of the teeth cut off the source of sepsis, the reactive powers of the patient were insufficient to cope with the eye infection until assisted by the vaccine.

The case shows the occasional therapeutical value of vaccines for treating lesions started by septic foci in the mouth, but the vaccine in this case would have proved of little value if the septic focus had been allowed to remain. It is highly probable that if the teeth had been removed as soon as the eye lesion was recognized the sight of the eye would have been saved.

Ionic medication is claimed by many practitioners to be of great value in the treatment of periodontal disease. My colleague, Mr. Norman Bennett, for whose opinion I have a great respect, has kindly contributed the following notes on this method of treatment.

Electro-therapeutic Treatment.

Basis of Therapeutic Action.—Electro-therapeutic treatment of periodontal disease depends upon the processes of cataphoresis and ionization separately or jointly. An electric current passed through a tissue or other substance has the power of transporting liquids, which may contain substances in solution, in the direction of the flow of current. The direction is from the positive to the negative pole, from the higher potential to the lower. In this way the tissue in the vicinity of the pole may be penetrated by an antiseptic drug to an extent impossible with mere application. This process is called cataphoresis. Another and probably more important effect of the passage of current therapeutically is the power of decomposing substances contained in solution, the "ions" migrating in opposite directions towards the positive and negative poles; penetration is thus produced as in cataphoresis, but instead of a soluble salt being forced unchanged into the tissues, the nascent metallic elements freshly liberated from their soluble salts are so used. This process is called ionization. It is exactly the same as the familiar process of gold-plating, in which a positive electrode of gold, a negative electrode of the article to be plated, such as German silver, and an electrolyte of gold cyanide are used; the gold salt is decomposed, the gold passes towards the negative pole and is there deposited, and the cyanogen reacts with the gold electrode and forms fresh cyanide, which goes into solution. Certain elements such as zinc, copper, silver (in fact, most of the metals), are called electro-positive, because they migrate from the positive to the negative pole. Most of the non-metallic elements, such as oxygen, chlorine, iodine, pass in the reverse direction, and are therefore called electro-negative. In the usual application of the electric current to the body in conjunction with a metallic salt, probably the two processes of cataphoresis and ionization occur coincidently.

Apparatus.—The current used is a weak constant current. Readers who desire a detailed description of apparatus should consult special works, but it may be well here to give a brief

description of the essential parts. The current may be obtained from the main (from dynamos) or from a battery of cells. In the former case resistance must be inserted to reduce the current to a low voltage, and certain precautions must be taken to insulate the chair from earth and the patient from his surroundings. A battery is equally convenient and absolutely safe, and is preferred by the writer. It should consist of a sufficient number of Leclanché or dry cells to give a maximum voltage of not more than forty volts. A current of this strength is too great for most purposes; it is therefore necessary to use a rheostat in circuit, so that a current of very low electro-motive force may be applied in the first instance and afterwards increased. A milliampere-meter should also be used in circuit, as it is necessary to know always what strength of current is being used. The active electrode may consist of a piece of platinum, zinc, or copper, flattened to form a small blunt spear-head point, and slightly curved. A convenient form for the indifferent electrode is a metal cylinder, which should be wrapped round with a thoroughly damp cloth and held in the patient's hand.

Technique. — It is of course to be presumed that before treatment is commenced calculus has been removed and the mouth generally got into as clean and healthy a condition as possible. It is not necessary, or in many cases possible, to remove all calculus as a preliminary to treatment; the process can be better completed during the course of treatment; indeed, small areas of marginal gingivitis that do not respond will often be found to be covering a small deposit of undiscovered calculus.

The drug that the writer has found most useful is a 5 per cent. aqueous solution of zinc choride; but salts of copper, such as the sulphate, or of silver, may be used.

A few contiguous teeth should be isolated and kept moderately dry by means of napkins, wool-rolls, or bibulous paper. Absolute dryness is not necessary, but too much moisture will not only dilute the drug unduly but also allow the current to pass superficially instead of through the tissues.

The current having been turned on and the rheostat control advanced a short distance, the electrode, previously wrapped round with a small quantity of cotton-wool, should be dipped in the solution and applied to the gum at least a quarter of an inch from the neck of a tooth ; it should then be slid carefully over the gum until the neck of the tooth is reached and passed down into the pocket round the tooth. The most important parts are the approximal pockets. It is usually convenient to treat about half a dozen teeth at a time, dealing first with the labial or buccal aspects and the approximal pockets, and completing on the lingual aspect. The amount of current registered with any given voltage varies inversely with the resistance of the body. This resistance itself varies with different patients and with the same person at different times. The amount of current that can be tolerated is usually from one to five milliamperes. It will usually be found that a current of one milliampere is not felt at all ; more than five cannot usually be borne, except perhaps in the molar region. A current of three milliamperes is usually sufficient for therapeutic effect, and tolerable to the patient. Of course, the character of the patient and of the teeth are determining factors.

A current of low electro-motive force having been tried at the outset, it can be gradually raised by moving the rheostat control until sufficient is obtained. It should be remembered that most pain will be felt when the needle is first applied (the "make" of the current), less when it is broken, and least of all during the passage of the current, so long as the electrode is stationary. That is why the needle should, every time it has been dipped in the solution, be first applied to the gums and then moved to the tooth. If a large drop of liquid is hanging on the needle a considerable amount of pain may be felt at the first contact. Care should be taken to move the needle smoothly from one tooth to the next without making contact or even altering the area of contact more than necessary, because the current is felt more severely when passed through a small area than a large. When the teeth are very sensitive it is sometimes necessary to apply the needle first and then

gradually turn the current on from zero; in this way pain may be greatly minimized.

By taking groups of teeth in succession the whole mouth may be treated in about three-quarters of an hour, but it is often desirable to spend the greater part of the time over the most seriously affected teeth, three or four minutes being devoted to a single tooth. The only visible sign noticeable is a slight whitening of the edge of the gum, and a kind of white deposit spreading from the edge. It is only very rarely that any sensation is felt in the hand of the patient holding the negative electrode. It is, however, desirable that rings should be removed.

The application should be repeated every second or third day for a fortnight, then less frequently until about ten or a dozen applications extending over five or six weeks have been made. In this period it will be possible to judge results, and if considerable improvement cannot be obtained in that time or even less, it is not likely to be gained at all.

Efficiency of the Method.—In discussing the efficiency of ionization it is necessary to have clearly in mind what is being attempted. In the opinion of the writer there are in most cases of periodontal disease a constitutional factor and a local factor, the latter being a bacterial infection of the pockets, the gum margin, and the alveolus. In some cases the former predominates, and in others the latter. Inasmuch as the rationale of the treatment consists mainly in a more perfect sterilization of the pockets than can be obtained in other ways, it is obviously in the infective cases that it will be likely to be most useful, and especially in early cases. On the other hand, a large number of cases of periodontal disease exhibit as their cardinal features a clean mouth, no marginal gingivitis, anæmic gums, considerable absorption of bone, and only shallow pockets, because the gums have receded *pari passu* with the absorption of bone. These are the cases that are thought usually to be associated with absorption of bacterial products into the blood-stream, rather than by the intestinal tract, as when there is a marked flow of pus from the pockets: they are the cases generally connected with arthritic affections. In these patients it cannot be expected that a sterilizing

effect will be so valuable. The writer believes, however, that the treatment is beneficial. It is more than likely that the current has a stimulating effect on the metabolism of the tissues, and there is also a secondary result that is much appreciated by patients. It is well known that in the kind of case now being considered far more pain and discomfort are occasioned by the disease than in the more septic cases. The necks of the teeth become acutely tender, so that meals become a source of misery. The writer has satisfied himself that if a current of low electromotive force is used for the first few sittings and gradually increased, this sensitiveness can be much reduced and even abolished, and that in some cases the cure remains good for a very long time.

In the class of cases first referred to—those in which there is marked gingivitis, deep pockets, and much pus—it is obvious that the treatment cannot be expected to produce infallibly a permanent cure. The pockets may be sterilized, but will probably become reinfected, and the whole train of consequences will be reproduced unless further steps are taken.

In early cases the writer considers that ionization affords the best means of sterilizing the gum margins and shallow pockets, and if accompanied and followed by reasonable prophylactic measures on the part of the patient effects a practical cure.

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